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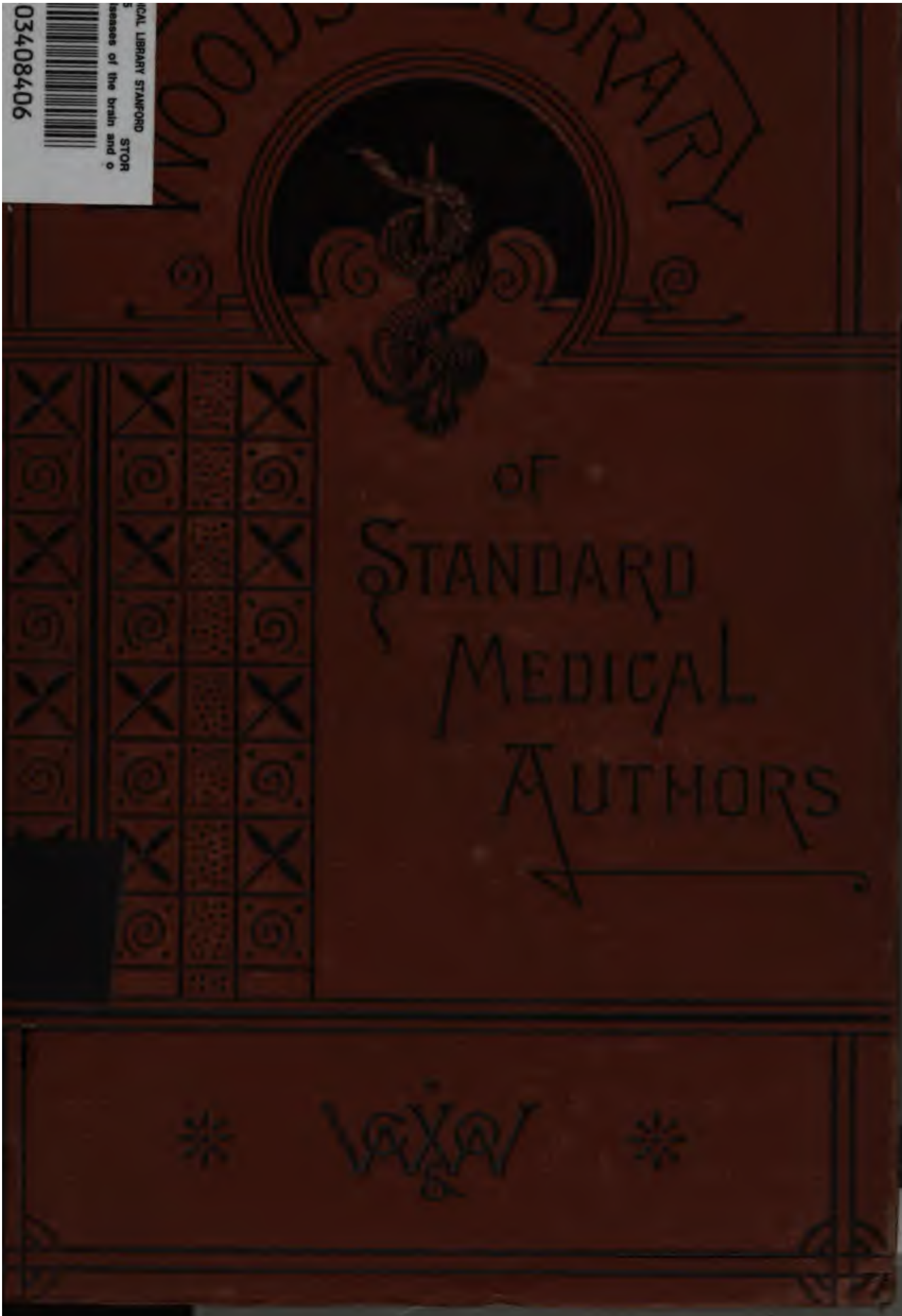

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DIAGNOSIS

OF

DISEASES OF THE BRAIN

AND OF THE

SPINAL CORD

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BY

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PREFACE.

Many readers of my "Diagnosis of Diseases of the Spinal Cord" have asked me for a book, similar in scope and method, dealing with the symptoms and methods of diagnosis of Diseases of the Brain. In response to that request, I publish these Lectures, delivered at University College Hospital.

I have left the Lectures very much in the form in which they were delivered, believing that thus they will be most useful. The account of the diagnosis of the nature of the lesion, contained in the concluding Lectures, is intended rather to impress on the student the methods of diagnosis and the most important distinctions between the various diseases, than to furnish an exhaustive description of these distinctions. At the same time, I believe that those given will enable a diagnosis to be made in the majority of the cases that the student or practitioner is likely to encounter.

Queen Anne Street, London,
June, 1885.

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THE

DIAGNOSIS

OF

DISEASES OF THE BRAIN.

LECTURE I.

INTRODUCTION—MEDICAL ANATOMY OF THE BRAIN; CORTEX, MOTOR PATH, SENSORY PATH.

GENTLEMEN,—The subject of the diagnosis of diseases of the brain certainly transcends in complexity, and perhaps exceeds in interest, all other problems in practical medicine. In nerve-elements the involution of energy reaches the highest degree known to us, and in the chief organ of the nervous system these elements, vast in number, various in character, are arranged in what seems to us extreme complexity. The number of the nerve-cells of the brain is not known, and if it were, our minds would be powerless to grasp the extent of the array. The cortex alone has been estimated to contain eight hundred millions of cells. Of all the elements that compose the brain, not one is isolated. Each is connected with others, and their anatomical connection subserves a corresponding functional association. A change in the functional state of one involves a change in the functional state of others; and change is constant, rest unknown.

Complex as the arrangement of the elements of the brain appears to us to be, it may be found, with fuller knowledge, that the principles of the plan are comparatively simple. The variety in form of the nerve-elements is small, if we consider how numerous they are. The fibres present little variation. The cells, even when the element of size is taken into consideration, may probably be classed in not more than a dozen varieties. It is the arrangement and connection of these elements that constitutes the complexity of structure of the brain, and subserves its complexity of function. At the same time we must remember that the varieties of form give us no clue to the differences that may exist among the elements of which nervous tissues consist. Neither microscopical examination nor chemical analysis can penetrate beyond the coarsest outlines of the constitution of living matter. It is probably within the facts to assert that each nerve-cell contains as many material atoms as the brain contains nerve-cells. Even the molecules, formed by the grouping of these atoms, are beyond the reach of any means of scrutiny we possess, or of which we have, at present, any promise. The fundamental diversity that must exist, where we can discern only uniformity, is shown to us by the action of poisons, and in the effects of disease. One poison acts on one part of the nervous system, and leaves uninfluenced another part, which a second poison affects alone. We are accustomed to explain this by the difference in the chemical constitution of the poison; but it is evidence of an equal difference in the nerve-elements, that are, as we say, "selected" by it. The selection is no matter of arbitrary choice. It is the result of the nature of the nerve-elements themselves; the expression of differences between them, revealed by such effects alone. This consideration renders many effects of disease more intelligible, though it may not lessen their mystery.

Certain leading facts regarding the structure of the brain must be clearly recognized, if we would understand what is known of the principles of the diagnosis of its diseases. Our present knowledge of the cerebral structure is at once far more than we can use, and far less than we need. The researches of the last quarter of a century—of Lockhart Clarke, Broadbent, and others, in this country; of Stilling, Meynert, Wernicke, Flechsig, among the Germans; of Luys, Foville, and others, in France—have brought to light an immense number of facts, and have built up a large mass of knowledge, much of which has at present no practical application.

But if we attempt to select from the facts described by different investigators those that are of chief importance, we are at once met by a fresh difficulty. Many of the conclusions reached by different investigators do not correspond—are even contradictory. This is not surprising. The interlacing tracts of fibres baffle the scalpel, and even the microscope, when we attempt to trace their course. What, then, is to be our guide when investigators disagree? Fortunately we are not dependent only on simple dissection, or even microscopical examination. The selective action of disease affords invaluable help in the study of the structure of the nerve-centres. It was long ago discovered (by Waller) that if nerve-fibres are destroyed at a given point, they undergo degeneration beyond the lesion, and this degeneration extends along them as far as they continue simple fibres. The degeneration is only in one direction, and that is the direction of functional conduction; on the side of the lesion from which the fibre conducts there is no degeneration. By means of this "secondary degeneration" the course of some tracts of fibres may be traced with precision through the brain. Moreover, we have a second guide. A distinguished German investigator, Flechsig, has availed himself of the fact that different tracts of nerve-fibres in the brain acquire their white substance at different stages of fœtal and infantile life. He has shown, by a remarkable series of investigations, that we may learn as much of the course of fibres by studying them in their birth as in their death—in their development as in their decay. His discoveries have thrown new light on many difficult problems, and supply a trustworthy guide in discovering where the truth lies in other and contradictory descriptions.

The experimental researches on the functions of the brain, chiefly of Hitzig and Munk in Germany, and Ferrier in this country, have also given us vast help in diagnosis. Here also, however, we meet with contradictions; and here also more has been ascertained than we can at present apply. Our guide in this department must be the facts of clinical and pathological observation. We must beware of applying wholesale to the human brain the conclusions derived from experiments on animals. The latter are of value to us only as indications for observation on man, and by enabling us to give a fuller interpretation to the facts we learn by our study of disease during life and after death. Some of the experimental facts have at present received no confirmation, and on some points we especially need information, which, from their nature, experiment cannot give.

Before we enter on those details regarding the structure and functions of the brain that are of chief importance for diagnosis, I must remind you of certain important elementary facts. We speak of nerve-cells and nerve-fibres as if they were merely connected structures, essentially distinct. They are not really so. The axis-cylinder of each nerve-fibre is the prolonged process of a nerve-cell, sharing all changes of nutrition that the nerve-cell undergoes, suffering with it when the cell is damaged. This is the secret of the secondary degeneration. If a fibre, or part of a fibre, is cut off from its parent cell, it degenerates; the part still in connection with the cell does not degenerate. If the cell is destroyed, the whole fibre perishes. Although I have said that every fibre is a nerve-cell process, I need hardly tell you that the fact is not proved. It never can be proved by observation. But the relation can be observed of some cells in various parts of the nervous system; the contrary has never been observed; and we may therefore infer, with considerable probability, that the fact is true of all nerve-fibres. We do not know whether any nerve-fibres unite directly the undivided processes of two nerve-cells. It is highly probable that, as a rule, they do not, because we usually find that only one process of a cell becomes directly an axis-cylinder; the other processes divide and ramify in a branching network of the finest nerve-fibrillæ. This structure is probably intermediate between adjacent nerve-cells, and in it the nerve-fibres may end, that are the prolonged processes of other and sometimes distant nerve-cells.

Another preliminary consideration is of a different nature. It is necessary, in speaking of the functions of the brain, to use the term "centre." Remember that the word is employed in its physiological sense; not in a geometrical, or even in a topographical, sense. It indicates a combination of nerve-cells subserving a given function. Note, first, that this function may not be the only one subserved by the group of nerve-cells. In many groups the nerve-cells are very numerous; they are connected with each other by a ramifying plexus of fibrillæ, and are connected with other and distant groups of cells by nerve-fibres. Such a collection of cells may contain many centres, since only some cells are probably in action at the same time. The possible functional combinations in such a group are almost infinite, since the variations involve not only the number of cells in action, but the relative degree of their action. Only some of these are usually, perhaps ever, in functional activity. Lines of various resistance exist by which the functional combinations are determined. Education is largely the establishment of these lines of least resistance. Habit depends upon them. Although the finest, most complex mechanism of human contrivance is rude compared with the simplest arrangement of the brain, I may illustrate this variation of function by an object with which you are all familiar. Wherever large buildings are being erected, or extensive excavations made, you will see a steam crane. This machine, by a change in the relation of the different parts of the complex mechanism, may be made either to draw up a weight, to swing itself round, or to travel along a line of rails. The motive force is the same—the machinery is essentially the same; the difference in result depends on certain parts of the machinery being in or out of "gear." In the one machine are functional centres for progression, rotation, and turning a windlass.

Not only may one part of the brain contain many centres, but a single functional centre may consist of nerve-elements that are anatomically distant—even situated in different hemispheres. Cells may act together when the nerve-fibre that connects them is many inches long, as perfectly as if they are only a hundredth of an inch apart, just as the needles of two galvanometers in the same circuit are deflected at the same moment, judged by ordinary standards, whether they are distant a foot or a mile.

One more preliminary consideration. A familiar line asserts that "things are not what they seem," and although the statement is scarcely of the universal application that its form suggests, it is one that should not be forgotten when we study the arrangement of the nervous system. The course of nerve-fibres is often very different from that suggested by the course of the nerves in which they run, and the arrangement and connection of the elements of the brain is often not the same as that suggested by the coarse morphological relations of its parts. Nerve-fibres frequently take a course not only very different from that which might be anticipated, but mysteriously tortuous—I was going to say unnecessarily tortuous. Doubtless these irregularities have had their necessity; but this lies in the dim past of development, beyond our range of vision. The course of the fibres of the cranial nerves affords many illustrations of these anomalies. A comparatively simple instance is presented by the spinal part of the spinal accessory nerve, which arises from the same gray matter as the cervical nerves, is distributed to the same muscles, but has a course that takes it just within the entrance to the cranial cavity, although it has no business there, as far as we can see, to justify its ascent. The course of the fibres that subserve taste is a more elaborate instance of the same thing, and it has caused difficulties from which neither anatomical nor physiological investigation has yet cleared our knowledge.

I may safely assume, gentlemen, that you are familiar with the chief facts in the topographical anatomy of the brain, and amongst them with the names of the convolutious. But, as I shall have frequently to refer to these, I put before you a diagram on which the fissures and convolutions are shown and designated.

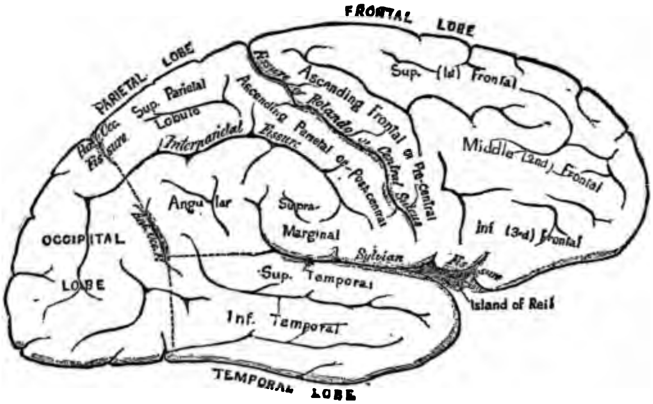

FIG. 1.—DIAGRAM OF THE CONVOLUTIONS AND FISSURES ON THE OUTER SURFACE OF THE RIGHT HEMISPHERE.

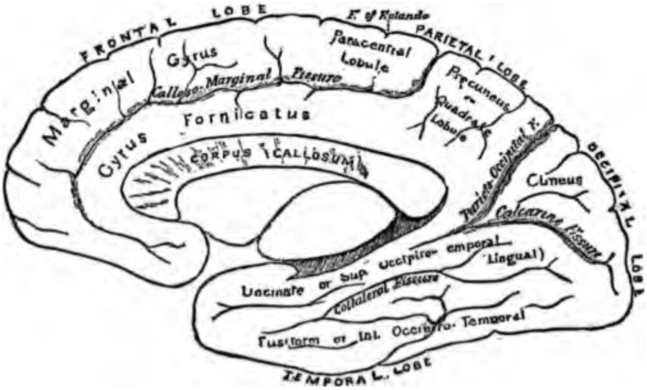

FIG. 2.—DIAGRAM OF THE CONVOLUTIONS AND FISSURES ON THE INNER (MEDIAL) SURFACE OF THE RIGHT HEMISPHERE.

I need only add one fact to those that are indicated on the diagrams. There is, as we shall see, an important difference in function between the ascending frontal and the three other frontal convolutions. Hence it is often convenient to speak of the latter without the former. We do so by terming the three antero-posterior convolutions, and corresponding inner surface, the "prefrontal lobe." Thus we mean by this term the portion of the brain anterior to the ascending frontal convolution. It is, like many others, an inaccurate word, but it has become current, and is convenient—indeed necessary.

The cortex of the cerebral hemisphere covers, as you know, a mass of white substance—the centrum ovale. The cortex consists of nerve-cells, with fibres connecting them, and proceeding from them to the white substance beneath. The cells vary much in form and size, and certain kinds of cells preponderate at different depths from the surface, thus enabling us to distinguish certain layers (see Fig. 3), which are, however, ill-defined, and differ somewhat in different parts of the cortex. Hence various descriptions of these layers have been given by different observers. At the surface is a narrow "*superficial* layer,"[1] with few nerve-elements, and beneath this is a layer of *small pyramidal cells* densely massed together. Below this is a thicker layer of *large pyramidal cells,* which are larger in the deeper than in the more superficial part of the layer. The apex of the pyramidal cells is turned towards the surface. Next comes a layer which contains still larger cells, some pyramidal, and others more irregular in form, and, from the resemblance of the latter to the cells of the anterior cornu of the spinal cord, this layer is termed the *ganglion-cell layer.* In the motor region of the cortex these cells are especially large, many of them exceeding in size any other cells in the nervous system. But between this layer and the last, or rather in the lowest part of the last, the large pyramidal layer, small round or angular bodies are rather numerous; and over a considerable part of the cortex, especially that in which the sensory functions seem chiefly developed, these increase in number, and constitute a well-marked *granule layer.* Beneath the ganglion-cell layer, and next the white substance of the hemisphere, is a layer in which *fusiform cells* preponderate.

The white substance consists of fibres passing in various directions. Some pass from one part of the cortex to another part in the same hemi-

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[1] The italics indicate the names commonly given to the layers.

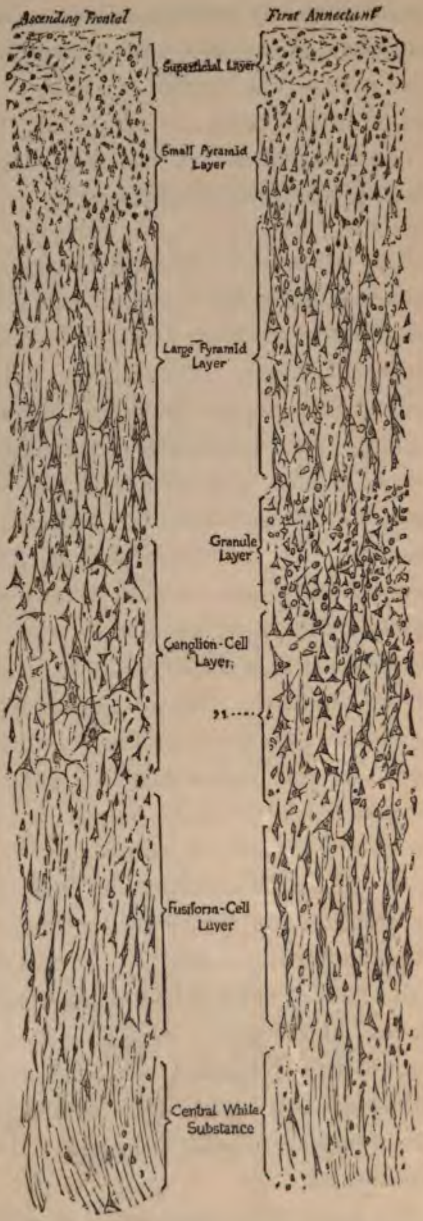

FIG. 3.—DIAGRAMMATIC SKETCH OF THE LAYERS OF THE CORTEX CEREBRI.

The drawings were made from sections of the ascending frontal and first annectant convolutions (the latter near the extremity of the parieto-occipital fissure). The sections were kindly furnished me, as representative of the so-called motor and sensory types, by Dr. Bevan Lewis, whose important investigations into the structure of the cortex are well known. In addition to the facts mentioned in the text, certain others may be noted. No layer is composed exclusively of one kind of cell; most forms are met with in each layer, but some are scarce and others abundant. Almost all the pyramidal cells have their apex turned towards the surface of the brain. The granule layer of the sensory type is formed at the expense of the two adjacent layers, the large pyramid layer and the ganglion-cell layer, and these two layers in the motor type merge one into the other to an even greater degree than the other layers. In the sensory type the very large ganglion cells of the motor type are absent. In the white substance nuclear bodies lie among the fibres, and near the gray cortex isolated cells occur, similar to those in the gray matter, becoming few as we pass more deeply into the white substance. Many fibres run through the gray substance towards the surface, and they are, to some extent, aggregated into bundles, partially separating the nerve-cells into columns. To avoid confusion these fibres have not been shown in the diagram.

sphere. Others pass inwards to the corpus callosum, and probably, through these, corresponding parts of the two hemispheres are connected, and brought into functional unity. Others, from almost all parts, converge to the internal capsule, and to the optic thalamus; while from the posterior and under part, fibres pass to the crus, and from the posterior part, to the corpora geniculata, quadrigemina, and optic tract.

In our survey of what may be termed the functional anatomy of the brain—the consideration of the structural arrangements that subserve special functions—we may pass next to the parts concerned in voluntary motion. As you already know, in only one part of the cortex can we trace a special relation to voluntary motion. This part consists of the two (so-called "central") convolutions that bound the fissure of Rolando, the ascending frontal and ascending parietal, with the expansion backwards of the latter in the "superior parietal lobule," and also with the medial aspect of these convolutions on the inner side of the hemisphere —the "paracentral lobule," and part of the "precuneus" (Fig. 2). As you have also learned in the class of physiology, voluntary motion is not uniformly related to this region. The leg is chiefly represented in the upper third of these convolutions, the arm in the middle third, the face in the upper part of the lower third, the tongue and lips in the lowest part of the ascending frontal (Fig. 4). Voluntary speech is related to

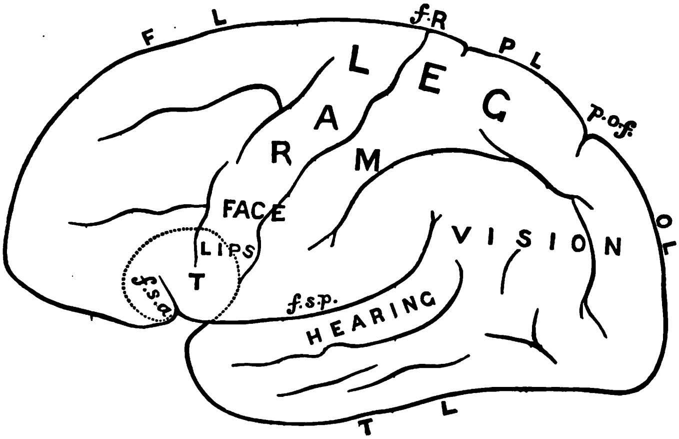

FIG. 4.—CORTICAL CENTRES: LEFT HEMISPHERE.

The position of the limb-centres is indicated by the letters composing their names. T, tongue-centre. The dotted line incloses the motor speech-centre. F L, frontal lobe; P L, parietal lobe O L, occipital lobe; T L, temporal lobe; *f.* R, fissure of Rolando; *f.s.a.*, anterior limb of the fissure of Sylvius; *f.s.p.*, posterior limb of the same fissure; *p.o.f.*, parieto-occipital fissure.

this centre for the tongue and lips, and to the adjacent part of the third (lowest) frontal convolution.

We do not know whether there is any sharp limitation between these limb-centres; probably there is not, at any rate between the centres for the arm and for the leg. In animals certain movements have been found specially excitable at certain points, and representations of the human brain are often given, to which these centres have been transferred. But clinical facts have not at present confirmed in man the existence of these limited centres for certain movements. In animals, moreover, centres are found on the posterior parts of the first and second frontal convolu-

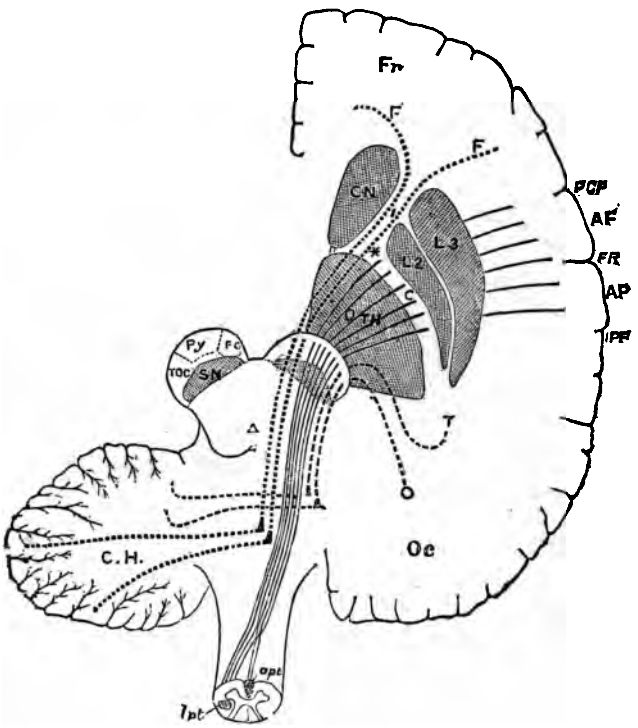

Fig. 5.—Diagram of the Course of the Motor Tract as shown in a diagrammatic horizontal Section through the Cerebral Hemisphere, Pons, and Medulla.

Fr, frontal lobe; Oc, occipital lobe; A F, ascending frontal; and A P, ascending parietal convolutions; *P C F*, precentral fissure in front of the ascending frontal convolution; *I P F*, interparietal fissure. A section of the crura is lettered on the left side: S N, substantia nigra; Py, region occupied by the pyramidal fibres (motor tract), which on the right are shown as continuous lines, converging in the white substance of the hemisphere, to pass through the posterior limb of I C, the internal capsule (the elbow of which is shown at *)—through the crus and pons, and to divide in the medulla into the decussating lateral pyramidal tract (*lpt*), and the non-decussating or anterior pyramidal tract (*apt*).

tions. These are not only unconfirmed so far as the human brain in concerned, but the balance of evidence is against their existence.[1]

This region of the cerebral cortex is certainly motor, but it is certainly also not exclusively motor. Its destruction by disease causes persistent paralysis, corresponding in distribution to the part destroyed; irritation of it causes convulsions, that begin in the limb corresponding to the part irritated. But destruction of these parts causes also some loss of sensation, chiefly in the extremity of the limb most paralyzed, and accompanied by an inability to recognize the position of the extremity, hand or foot—an inability which may be out of all proportion to the loss of cutaneous sensibility, and even, strangely enough, may exist alone. Of this peculiar loss I shall have more to say hereafter. An-

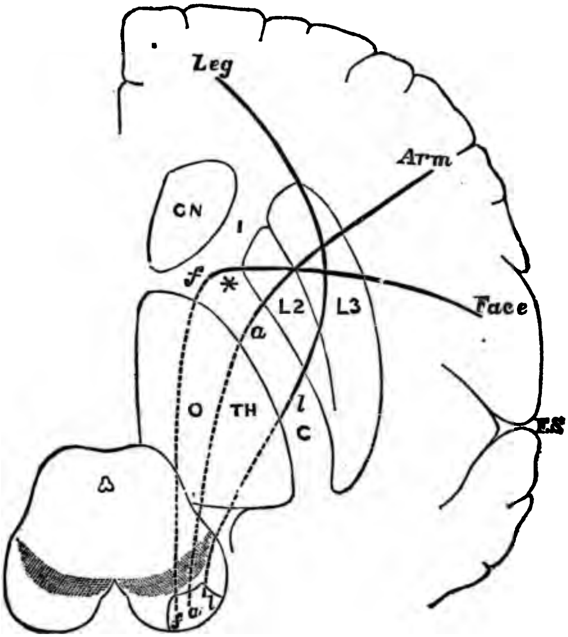

FIG. 6.—DIAGRAM TO SHOW THE RELATIVE POSITION OF THE SEVERAL MOTOR TRACTS IN THEIR COURSE FROM THE CORTEX TO THE CRUS.

The section through the convolutions is vertical; that through the internal capsule, I C, horizontal; that through the crus is again vertical. C N, caudate nucleus; O TH, optic thalamus; L 2 and L 3, the middle and outer parts of the lenticular nucleus; *f*, *a*, *l*, face, arm, and leg fibres. The words in italics indicate the corresponding cortical centres.

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[1] Schäfer and Horsley find, in monkeys, centres for the trunk muscles in the highest part of the motor region (especially on the inner aspect), and in the posterior part of the first frontal convolution. Ferrier found centres for the movement of the head and eyes on the first and second frontal.

other fact of similar significance is that convulsions produced by disease in this region often commence by a sensory "aura."

From this motor region of the cortex, fibres pass down to the spinal cord, and conduct the motor "impulses," as we call them, from the motor mechanism of the convolutions to the motor mechanism of the gray matter of the cord. Leaving the cortex, these fibres pass through the white substance of the hemisphere, converging to the "internal capsule," which, as you know, lies between the lenticular nucleus (or extraventricular part of the corpus striatum) on the outside, and the caudate nucleus, and optic thalamus, on the inside. Thence the fibres pass into the crus, through the anterior part of the pons, and constitute the anterior pyramids of the medulla. Crossing for the most part in the decussation of the pyramids, they continue down the cord, where we have seen them as the pyramidal tracts,[1] lateral and anterior. This is a long course; the longest of any fibres of the central nervous system. It was formerly thought that this course was broken in the gray matter of the corpus striatum; but this seems to be an error. The cells of the corpus striatum have no direct relation to voluntary movement; the motor tract passes in an unbroken course from the cortex down to the gray matter from which the motor nerves directly proceed.

Certain points in the arrangement of the fibres must be noticed. They do not constitute the whole of the internal capsule. In horizontal section (Fig. 5) this capsule seems to consist of two oblique parts, which join at an angle; the anterior segment, or "limb," as it is termed, lies outside the body of the caudate nucleus, the posterior outside the optic thalamus, and the angle or "elbow" (the "Knie" of the Germans) lies between the thalamus behind, and body of the caudate nucleus in front. The motor fibres occupy only the elbow and the anterior two-thirds of the posterior segment. The posterior third of this, as we shall see, contains sensory fibres, and the anterior segment contains fibres of uncertain function, which pass down from the frontal lobe to the cerebellum. We shall return to these in a subsequent lecture. The fibres from each limb-centre in the cortex keep together in the internal capsule, but the several groups exchange their vertical relation for one that is antero-posterior (Fig. 6); the fibres for the face, and probably for the tongue, occupy the elbow of the capsule, then come the fibres for the arm, and, most posterior, next to the sensory fibres, come the fibres for the leg.

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[1] "Diagnosis of Diseases of the Spinal Cord," 3d Ed., p. 10.

From the internal capsule the fibres descend into the crus, lying in the anterior or lower division (crusta), and occupying the middle two-fifths of this region, but separated by some other fibres from the locus niger above them. To their inner side lie the fibres that formed the anterior limb of the internal capsule, and to their outer side is another bundle of fibres, of which I shall have more to say. The relative arrangement, which was vertical in the cortex, and antero-posterior in the capsule, here becomes transverse (see Fig. 6), the fibres for the face lying to the inner side, and those for the leg to the outer side, of the fibres for the arm. The fibres for the tongue probably lie close to those for the face. The latter two sets of fibres leave the others in the pons, and cross the middle line to the nuclei from which the facial and hypoglossal nerves proceed.

We know much less of the sensory path. Even in the spinal cord it is uncertain. The older theory (of Brown-Séquard), that sensation is chiefly conducted in the gray matter, is not disproved, but there is reason to believe that some sensation is conducted by the fibres of the lateral column in front of the pyramidal tract, and by fibres of the posterior columns. In the medulla and pons the path probably passes up in the posterior half, above the "fillet," perhaps chiefly in the curious network of fibres called the "reticular formation." It has been conjectured that some sensation may be conducted through the cerebellum; but this seems, on the whole, improbable. The path passes up beneath the corpora quadrigemina, through the tegmentum of the crus above the locus niger, and enters the internal capsule, where we are able exactly to determine its position. It occupies, as I have already told you, the posterior third of the hinder limb of the capsule. These fibres were once thought to be the same as those which, in the crus, occupy the outer fifth of the crusta; but Flechsig has shown that this is an error. These outer crustal fibres radiate into the white substance of the occipital and temporo-sphenoidal lobes, and connect those lobes with the cerebellum. As these fibres leave the crus, their place is taken by sensory fibres from the tegmentum, which thus form the posterior part of the internal capsule. Hence you will perceive how uncertain is our knowledge of the greater part of the course of the sensory path. Between the posterior roots of the spinal nerves and the internal capsule we have no definite facts regarding its position. We know only that it crosses the middle line in the spinal cord, not far above the level at which the nerves enter, and that it passes up the pons on the same side as that which it occupied in the cord. In the upper part of the pons the path from the fifth nerve—from the face, etc.—joins it, so that the posterior part of the internal capsule conducts sensation from the whole of the opposite half of the body and head, skin and mucous membranes, as far as the middle line. Moreover, the facts of pathology show that the path of cutaneous sensibility is here contiguous to the path of special sensibility from the organs of special sense—of taste, hearing, smelling, vision—which receive sensory impressions from the opposite side. Hence Charcot has termed this region the "sensory crossway." I say "from the opposite side," rather than "from the nerve of the opposite side," because in the case of vision, the impressions that pass by this sensory region are those which come from the half of each field of vision corresponding to the side from which the other sensory impressions come. We shall have presently to consider this point more fully.

The ultimate destination of the sensory path is still to a large extent uncertain. Some fibres enter the optic thalamus, but their function is unknown, as there is not at present any evidence to show that impressions which influence consciousness pass through the thalamus, except, perhaps, in the case of visual impressions. The sensory fibres pass into the white substance of the hemisphere, and go towards the parietal and central ("motor") region; roughly speaking, towards that part of the cortex that lies under the parietal bone (Flechsig). We have already seen that disease of the motor cortex often causes impairment of tactile sensibility; and that when convulsions are caused by disease of this part of the surface, they often begin with a sensory aura. Thus clinical evidence, as well as the facts of anatomy, points to the outer part of the hemisphere in the middle region as being the part concerned in sensation. A case which has been recorded by Demange,[1] shows that the indication is correct, and places the conclusion beyond doubt. This case is so important that I must mention its leading facts: A woman, aged 72, after suffering for two weeks from tingling and numbness in the left arm and leg, was found to have almost complete loss of sensation of touch, pain, and temperature in the whole of the left side, face, and limbs; with amblyopia and loss of color-vision in the left eye. (We will consider subsequently the significance of this loss of sight.) The mucous membranes were not then anæsthetic, but a fortnight later were found to have lost sensibility, and the cutaneous anæsthesia was complete up to the middle line. Smell and taste were also lost on the left side, and the impairment of vision was greater. There was also considerable weakness in the left limbs; this gradually increased, and involved the face and the tongue. These symptoms continued until death, six months after the onset. An extensive area of softening was found, involving a large part of the outer surface of the right hemisphere and subjacent white substance; the inner surface of the hemisphere, central ganglia, and internal capsule were unaffected. The precise area of cortical damage was—lower two-thirds of the ascending frontal and ascending parietal, with the posterior extremities of the middle and lower frontal; island of Reil, inferior parietal lobule, superior parietal lobule adjacent to the intraparietal sulcus, the angular gyrus, and the whole of the occipital and temporo-sphenoidal lobes on the outer surface. The softening of the inferior parietal and angular convolutions seemed of rather older date than that elsewhere.

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[1] "Revue de Méd." May, 1883, p. 391.

This case affords a crucial demonstration of the correctness of the indications afforded by the anatomical researches of Flechsig. We shall have to return to it again when we consider the central relations of vision.

LECTURE II.

MEDICAL ANATOMY OF THE BRAIN (*Continued*): NERVES OF SPECIAL SENSE—OTHER CRANIAL NERVES.

Gentlemen,—In our survey of those points in the anatomy of the brain that are of chief medical importance, it may perhaps be most convenient to study next the central relations of the cranial nerves. We will consider first, however, those nerves that subserve the special senses, and will begin with that which is most difficult, of which we have most knowledge, and yet (perhaps therefore) see most clearly how imperfect our knowledge is—the nerve of sight. At the optic chiasma these nerves undergo a partial decussation, rather more than half the fibres crossing. Fifteen years ago this semi-decussation was regarded as satisfactorily proved; but in this questioning age few doctrines seem unassailable, and it has been maintained that there is a total decussation. The result of the discussion that has taken place is to show that the old doctrine is correct, and to place it upon a firmer basis. In some animals there is a total decussation; but this occurs in those creatures in which the eyes are so placed that they never act together, and in which the fields of vision are entirely separate. In proportion as the two eyes are used together, and the fields of vision correspond, the fibres cross, so as to bring the corresponding regions of each retina into relation with one cerebral hemisphere. In man the inner half of each field of vision is smaller than the outer half, because it is limited by the projecting nose. Although the two halves of the *retina* are of equal size, the sensitiveness of the outer, temporal half (which receives rays from the inner, nasal half of the field of vision) does not extend so far towards the front as does that of the inner, nasal half. This is shown by examining the field—first when the eye is directed forwards; secondly, when it is directed outwards. The limit of the nasal half of the field is nearly the same in each case, although when the eye is directed outwards, the nose no longer cuts off the rays. Doubtless the structure of the retina corresponds to this; its temporal half contains fewer nerve-elements than does the nasal half, and from this half fewer nerve-fibres proceed. The fibres that decussate are those from the nasal half of each retina, and hence the fibres that cross are more numerous than those that do not cross. If the fibres from the whole of one retina have degenerated, the opposite optic tract shrinks in size more than the optic

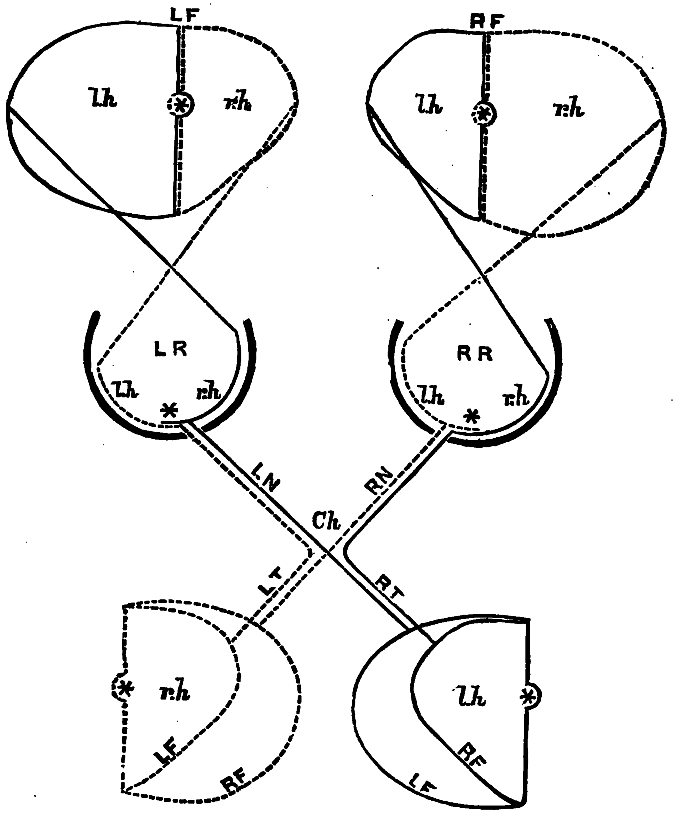

FIG. 7.—DIAGRAM OF THE RELATION OF THE FIELD OF VISION, RETINA, AND OPTIC TRACT ON EACH SIDE.

R F, L F, right and left fields—the asterisk is at the fixing point; R R, L R, right and left retina—the asterisk is at the macula lutea; *l.h*, *r.h*, left half and right half of each retina, receiving rays from the opposite half of the field; R N, L N, right and left optic nerves; *Ch*, chiasma; R T, L T, right and left optic tracts; below, the halves of the fields from which impressions pass by each optic tract are superimposed.

tract on the same side. Disease of one optic tract, right or left, therefore, arrests the conduction from the same-named half of each retina, and causes loss of vision in the opposite half of each field—"hemiopia," or "hemianopsia," as it is termed. (Fig. 8.)

The division between the two half-fields is usually in, or very near, the vertical line passing through the fixing point (corresponding to the macula lutea of the retina). Sometimes it passes through the fixing point; more often it diverges and passes round the fixing point, a little distance from it, to return again to the middle line, and this whichever side is blind. The balance of evidence at present collected seems to show that this difference does not depend on the seat of the disease causing the hemiopia; and it can therefore only be explained by individual differences in the decussation. In many individuals, it would seem, fibres from a small area around the centre of the macula pass by each optic tract; while in some persons, each tract contains fibres from only one-half of the macula region, as it does from the rest of the retina. Another form of variation seems to show also that there are considerable differences in the character of the decussation. Above and below the fixing point, the line of division is also often irregular, sometimes, for instance, sloping off to one side above, and to the other side below, the two fields corresponding closely. In an extreme example of this, such as is shown in Fig. 9, the division becomes irregularly

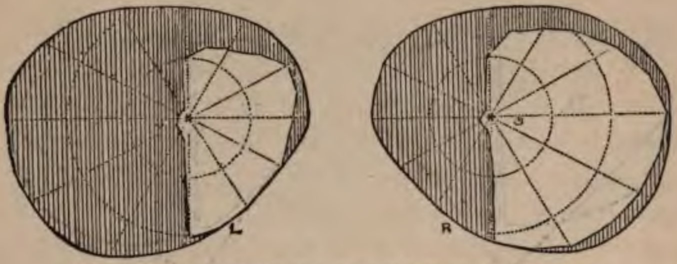

FIG. 8.—FIELD OF VISION IN A CASE OF LEFT-SIDED HEMIOPIA.

The shading represents the blind part; the oval outline of each figure is the average normal field. The asterisk is the fixing point. The small circle represents the blind spot.

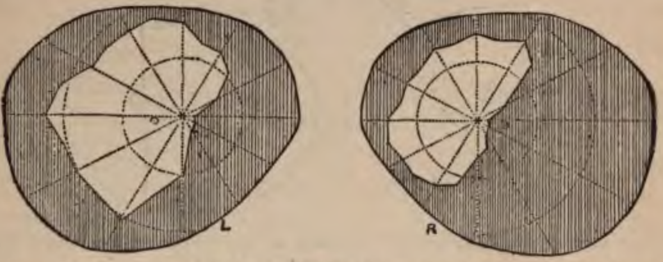

FIG. 9.—RIGHT HEMIOPIA FROM CEREBRAL DISEASE, SHOWING AN IRREGULAR OBLIQUE LINE OF DIVISION, PROBABLY DUE TO AN UNUSUAL FORM OF DECUSSATION.

oblique instead of vertical. It is impossible to explain this, except on the theory of variations in the decussation. Some facts suggest that there are similar variations in some other decussations in the nervous system, especially in that of the sensory tract from the skin.

The visual path passes in the optic tract, by the corpora geniculata, through the white substance of the hemispheres, to the cortex of the occipital lobe on the outer surface. If it is destroyed anywhere in this course, the result is hemiopia of the same character. Disease of the cortex of the occipital lobe has a like effect. There is no complementary decussation at the corpora quadrigemina, as has been fancied. Whether the fibres from the tract to the cortex are interrupted by gray matter, or are continuous, we do not know. Fibres from the tract enter the external corpus geniculatum, the optic thalamus, the corpora quadrigemina. Fibres from these pass into the white substance of the hemisphere, and the gray matter of these bodies *may* be interposed in the optic path. It is probable, however, that the fibres that enter the corpora-quadrigemina substance only guide the movements of the eyeball. On the other hand, some fibres from the tract pass directly into the hemisphere, and these may continue the visual path, or part of it, directly to the cortex.

The experiments of Munk lead him to believe that in dogs the anterior half of the half-vision centre of the occipital lobe subserves the upper quarter of the retina (and therefore the lower quadrant of the field); and the posterior half of the centre subserves the lower quarter of the retina (and therefore the upper quadrant of the field). The frequency of partial hemiopia in man, *e. g.*, the loss of a lower or upper quadrant, makes it certain that there must be an arrangement of the fibres and a central representation on a system similar to that in the dog, although whether the correspondence holds good in the details of the plan we do not yet know.

But the central relations of vision are still more complex. The subjects of that strange disease, hysteria, sometimes suffer from hemianæsthesia and loss of the special senses on the anæsthetic side, together with —not hemiopia—but what is termed "crossed amblyopia," dimness of sight of the eye on the anæsthetic side, peripheral constriction of the field of vision, and often loss of color-vision. There is usually also a far slighter limitation of the field of the other eye. The curious symptoms of hysteria probably depend on some morbid action in high cortical centres. They may teach us much of the functions of the brain, much of the association of its elements on which those functions depend; although they furnish no indication of the coarser anatomical relations. The occurrence of this amblyopia shows that there must be a functional centre, capable of being inhibited, in which is represented chiefly the whole field of one eye, not the half-fields of both eyes. It does not, however, show that this field is situated in one hemisphere, since the half-vision centres in the two hemispheres might be so connected as to be susceptible of such combined partial inhibition as would produce the symptoms. But the experiments of Ferrier have shown that lesions of the angular gyrus in animals will cause dimness of sight of the opposite eye, which rapidly passes away. In rare cases in man, in which there is certainly organic disease, similar crossed amblyopia has been observed. I have seen a few instances of this, and several others have been recorded. Moreover, the case I have quoted (page 14) is proof that such amblyopia may result from disease of the outer surface of the hemisphere. It will be noted that in this case the lesion involved the occipital lobe, and should have caused, therefore, hemiopia also, recognizable in the eye on the side of the lesion.[1] But the pathological appearances suggested that the disease of the occipital lobe was later than that of the angular gyrus; and such hemiopia might well have escaped detection during the later stages of the patient's illness. The original report of the case gives no indication that it was sought for.

Hence it is in a high degree probable that (as Ferrier has suggested) in or near the angular gyrus there exists a visual centre, higher than the half-vision centre, in which the whole of the opposite field is represented. For this relation there must be a connection between this centre on one side and both occipital lobes, that with the opposite occipital lobe being probably by means of the fibres of the corpus callosum. Indeed, the arrangement is probably even more complex. The crossed amblyopia is accompanied by a much slighter restriction of the field of the other eye, *i.e.*, on the same side as the lesion. Hence, in each higher visual centre both fields must be represented, that of the opposite eye, however, far more extensively than that of the eye on the same side.

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[1] I have recorded in "Medical Ophthalmoscopy" (2d Ed., Case 30, p. 311) a case in which total blindness of the left eye, with left hemiopia in the right, accompanied left hemiplegia—all due, no doubt, to an extensive lesion of the right hemisphere.

Another difference may be traced between this and the half-vision centre. A permanent lesion of the latter causes permanent hemiopia; but the crossed amblyopia soon lessens, and before long becomes slight. This is probably by the substitutionary action of the centre of the opposite hemisphere, since in each, as we have seen, both fields are represented. Lastly, a lesion of the higher centre, whatever its exact seat or nature, seems always to cause the same form of impairment (amblyopia, restricted fields, loss of color-vision), which differs in degree, but not in form. A partial lesion seems to lower the function as a whole—an indication that the function is diffuse,—and this is probably a characteristic of all the higher cortical centres.

Our knowledge of the central relations of the olfactory nerve is much less extensive. The only indication afforded by experiment is that there is a cortical centre for smell at the anterior extremity of the uncinate gyrus on the inner surface of the hemisphere (Ferrier). Towards this some of the fibres of the olfactory nerve seem to pass directly. Moreover, disease adjacent to these fibres has caused loss of smell on the same side as the lesion. On the other hand, disease involving the "sensory crossway" has impaired smell on the side opposite to the lesion. Hence, it seems probable that the olfactory path passes first to a cortical centre in the same hemisphere, and thence to the other hemisphere, although where it crosses, and what is its ultimate cortical destination, we do not know, except that the latter is on the outer surface of the hemisphere. This is shown by the case described on p. 14.

The auditory nerves pass to nuclei situated at the junction of the pons and medulla oblongata. Their exact position we shall consider presently. They have an extensive connection with the cerebellum, to which, indeed, some fibres of the auditory nerves seem to pass directly. You doubtless remember that one part of the auditory nerve (from the semicircular canals) has nothing to do with hearing, but conveys information, as to the position of the body, to the centre for equilibration, probably situated in the cerebellum. The auditory path to the hemispheres probably passes up the pons, crossing early, and, in the crus, occupies the superior portion of the tegmentum. Some have thought that it does not pass up the pons; but passes through the cerebellum; but this is, on the whole, improbable. Its course in the cerebrum is by the "sensory crossway," through the white substance, to the first temporo-sphenoidal convolution, which all evidence shows to be the cortical centre for hearing, each centre receiving impressions from the auditory nerve on the opposite side.

Of the path of taste, we know only that it passes by the sensory crossway; of its cortical centre we know nothing. Strangely enough, even the nerve of taste is still somewhat uncertain. It is commonly supposed that the glosso-pharyngeal is the nerve of taste of the back of the tongue, and that the chorda tympani of the facial, which is certainly the nerve of taste for the front of the tongue (and probably comes from the fifth by the Vidian nerve, that passes from the spheno-palatine ganglion to the facial), may ultimately be derived also from the glosso-pharyngeal. But I believe that it will be found that taste-impressions reach the brain solely by the roots of the fifth nerve, and that the doctrine that the roots of the glosso-pharyngeal nerve have anything to do with taste is a curious physiological myth, due to too wide an induction from certain anatomical facts, and from dubious experiments on animals. Some time ago[1] I published a case in which there was an isolated palsy of one fifth nerve, motor and sensory portions, due certainly to disease of the root at the surface of the pons, in which taste was entirely lost on that side, not only at the front of the tongue, but also at the back, on the soft palate, and on the palatine arches—lost to every form of stimulation, whether by sapid substances or by the voltaic current. The symptoms in this case are permanent, and I have repeatedly demonstrated the loss to those who have attended my practice at the Queen Square Hospital. Since the publication of that case I have met with only two other cases in which there was intracranial disease of the fifth without evidence of any interference with the nerves of the medulla, and in each of these there was the same absolute loss, at the back as well as at the front, demonstrable with ease, and most striking by comparison with the other side. It is possible that the nerve-fibres for taste on the back of the tongue may be *distributed* with the glosso-pharyngeal, reaching them from the otic ganglion of the fifth by the small petrosal nerve and tympanic plexus. This explains the remarkable fact, pointed out by Urbantschitsch, and which I have several times observed, that taste may be lost on the back as well as the front of the tongue, in consequence of caries of the walls of the tympanum. This course seems, I confess, strangely circuitous, but it is scarcely more circuitous than that which is certainly taken by the taste-fibres of the front of the tongue. If this arrangement is ultimately proved beyond question, it will be of much interest, as bringing the sense of taste into the functions of one nerve, and that one the nerve that is so intimately associated with the other special senses of smell, sight, and hearing.

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[1] "Journal of Physiology," vol. iii., p. 229.

We may conveniently consider next the arrangement of the nuclei from which the other cranial nerves arise. These are situated in the gray matter that surrounds the highest part of the central canal of the spinal cord, the continuation of this canal between the fourth and third ventricles, and that which lies beneath the floor of the fourth ventricle. The nuclei are columns of nerve-cells, small in transverse section, but prolonged in the direction of the axis of the medulla. (See Fig. 10.) The upper part of the hypoglossal nucleus lies close to the middle line in the point of the calamus scriptorius ; lower down it lies on each side of the middle line in front of the central canal. In corresponding position behind the canal is the column of cells of the vago-accessorial nucleus. In the calamus this lies outside the hypoglossal, and gives origin to the pneumogastric ; while below, part of the spinal accessory springs from it. The spinal part of the accessory consists only of spinal fibres that rise into a temporary companionship with a higher nerve, but have the same origin, and subside to the same distribution, as the other spinal nerves of the same level. Note, however, the proximity of the nuclei of the hypoglossal and accessory nerves; both supply muscles that act together in articulation. Moreover, the muscle that raises the palate and shuts off the posterior nares in articulation is certainly supplied from one of the nerves of the medulla, probably from either the glosso-pharyngeal or the spinal accessory. All these three parts—tongue, palate and vocal cord—are paralyzed together from disease at the surface of the medulla, damaging the roots of the nerves. All these parts are paralyzed together in degeneration of these nuclei, with the addition also of the lips, constituting the chronic form of "labio-glosso-laryngeal paralysis," or "bulbar paralysis." Why the lips are also affected we shall see presently. The pneumogastric nucleus in the floor of the ventricle corresponds nearly in position to the respiratory centre, and no doubt also to the cardiac centre. Its upper and outer limits are not definable, since it blends with a more diffuse area of delicate gray matter that extends as far as the auditory nucleus. Outside, and rather in front of the upper part of the pneumogastric nucleus, is the small column of nerve-cells from which the glosso-pharyngeal nerve arises.

The auditory nucleus is situated at the level of the auditory striæ, and chiefly in the outer part of the floor of the ventricle, just over the commencement of the inferior cerebellar peduncle, but it extends inwards almost to the middle line. It is a double nucleus, consisting of inner and outer parts. Fibres are said to pass from it to the cerebellum, and some fibres of the nerve seem to turn aside, and joining those of the restiform body, pass directly to the cerebellum by its inferior peduncle. The significance of this connection with the cerebellum has been already mentioned.

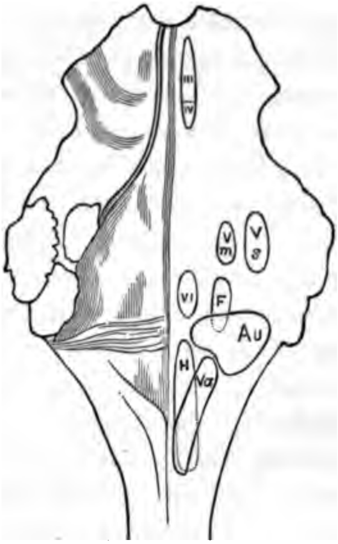

FIG. 10.—DIAGRAM OF THE RELATIVE POSITION OF THE NERVE NUCLEI BENEATH THE FLOOR OF THE FOURTH VENTRICLE.

III, third nerve nucleus; IV, fourth; V *s*, middle sensory nucleus of the fifth; V *m*, motor nucleus of fifth; VI, sixth; F, facial; Au, auditory; H, hypoglossal; V a, vago-accessorial nucleus, the upper part giving origin to the pneumogastric, the lower to the highest fibres of the spinal accessory. Where one nucleus lies beneath another, its outline is indicated by a dotted line.

Above the auditory striæ, under a prominence on the eminentia teres, close to the middle line, is the nucleus of the sixth nerve. It was once thought to be the common nucleus of the sixth and facial,—a strange combination, since these nerves have no functional association. The facial nerve ascends to this nucleus, forms a loop round it (some fibres, indeed, go through it), and then passes downwards, forwards, and outwards, to a column of cells more deeply placed in the medulla than any other nucleus in the lower part. A rare case, in which there was total palsy of all the ocular nerves, and no paralysis of the facial, afforded me an opportunity of proving beyond doubt that the facial nerve has no real origin from the nucleus of the sixth. All the cells of this nucleus were degenerated, but the fibres of the facial were perfectly healthy, and could be traced unchanged through the nucleus. The cells of the true facial nucleus cannot be traced far below the level of the auditory striæ, but it is probable that some fibres of the facial nerve, those that innervate the orbicularis oris, descend to the level of the hypoglossal nucleus, and may even arise from this nucleus. The transverse muscle of the tongue and the orbicularis act together. Neither can contract or relax without the other. Try, yourselves, gentlemen, when you are alone with a looking-glass, and you will find that this is true. Both, moreover, suffer together in degeneration of the nuclei, while the other parts of the facial nerve escape.

The chief nucleus of the fifth nerve lies above the sixth, and at the outer part of the floor of the ventricle. The fibres of the nerve pass backwards and slightly inwards, and end in a sensory nucleus on the outer, and the motor nucleus on the inner side. This is, however, but one part of the sensory nucleus. Some fibres pass down the medulla, and can be traced as low as the commencement of the spinal cord. They have gray matter on their outer side, in which they probably end. No doubt these fibres bring sensory impressions from the tongue, etc., into relation with the motor nuclei for these parts. Moreover, the distribution of the fifth nerve to the skin joins that of the cervical nerves, both on the face and back of the head, and the continuity of the cutaneous distribution is no doubt subserved by a continuity of the gray matter, in which the lower root arises, with that from which the cervical nerves arise. Another group of fibres of the fifth passes up beneath the corpora quadrigemina, no doubt subserving the intimate connection between the fifth nerve and the organ of vision, of which so obtrusive an instance is presented in the photophobia of conjunctivitis.

The third nerve, entering the inner side of the crus, passes back to a column of nerve-cells beneath the aqueduct of Sylvius, not far from the middle line. The combined teaching of experiment (especially of Hensen and Voelcker) and of clinical observation shows that there are three centres in this column of nerve-cells, distinct at least in function and in pathological liability. The most forward of these is the centre for the ciliary muscle (accommodation); the second is the centre for the light reflex of the iris; the third, which occupies the greater part of the nucleus, is the centre for the external muscles supplied by the third nerve.

The fourth nerves differ in two respects from any other of the cranial nerves. First, they arise above the fourth ventricle, their origin being from the valve of Vieussens, but they curve round the lower part of the aqueduct of Sylvius to a column of nerve-cells that is really the lowest part of the third nerve nucleus. Secondly, alone of all the cranial nerves, the fourth nerves decussate between the surface attachment and the nucleus. But the whole of the fibres do not come from this nucleus. Some descend the pons, and probably arise from the nucleus of the sixth nerve. Indeed, there seems to be an extensive connection, by large nerve-fibres, the "posterior horizontal fibres," between the three nuclei for the nerves of the external ocular muscles (Flechsig). It is possible that some of these are fibres of the nerves which really pass by their proper nucleus, and arise from one of the other nuclei, and the visible decussation of the fibres of the fourth nerve may be the indication of an extensive decussation of these connecting fibres. Thus we can understand how the apparently simple arrangement of the nuclei, and their apparently strange separation in three distinct groups of nerve-cells, may cover an extensive and complex structural association, whereby one nucleus may give origin to fibres that run in several nerves, and the

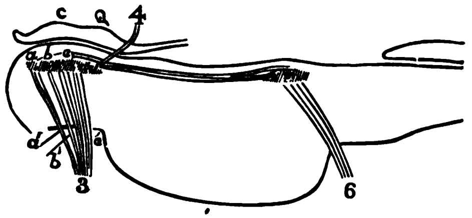

FIG. 11.—DIAGRAM OF LONGITUDINAL SECTION THROUGH THE PONS, SHOWING THE RELATION OF THE NUCLEI FOR THE OCULAR MUSCLES.

C Q, cor pora quadrigemina; 8, third nerve; III, its nucleus; 4, fourth nerve; IV, its nucleus, the posterior part of the third; 6, sixth nerve. The probable position of the centre and nerve fibres for accommodation is shown at *a* and *a′*; for the reflex action of iris at *b* and *b′*; for the external muscle at *c* and *c′*. The lines beneath the floor of the fourth ventricle indicate the fibres that connect the nuclei.

various nuclei may be blended into what is practically a series of centres very different from their apparent form. Thus, too, we can understand how these nuclei may govern the complex movements of the eyes, in which many muscles of both sides act together in the most perfect synchronism and exact gradation of effect.

The third, sixth, and hypoglossal nerves arise at the surface, near the middle line, and pass back to their nuclei between the middle line and the motor pyramidal tracts. The spinal accessory, pneumogastric, glosso-pharyngeal, and facial nerves arise on the surface at the outer part of the medulla, on the outer side of the pyramidal tracts, and pass inwards and backwards to their nuclei. The nuclei of the auditory nerve lie almost immediately above its origin, and one part of the nerve passes directly back to it, while the other curves round and above the outer nucleus to reach the inner nucleus.

From all these nuclei, paths, as yet only partially traced, ascend to the cerebral hemisphere. They certainly cross the middle line not far above the nuclei. The paths, motor and sensory, probably join, or at least run close to, the motor and sensory paths from the limbs. This, as we have seen, is certainly the case with the motor path of the face.

After leaving the brain, the nerves have a short course before entering the dura mater. Most arise in the posterior fossa. The nerves to the orbit and the fifth nerve leave the skull in the middle fossa, but as they leave the posterior fossa they pass into the dura mater. Remember that the sixth nerve has by far the longest course before it enters the dura mater, from the posterior border of the pons to near its anterior border, and that before it enters the wall of the cavernous sinus it passes very near the fifth nerve. Remember also that in its course over the convexity of the pons it readily suffers from pressure, if there is any cause of pressure beneath the tentorium. For this reason, paralysis of both sixth nerves is a very common symptom in disease of this region.

LECTURE III.

MEDICAL ANATOMY OF THE BRAIN (*Continued*): CONNECTION OF CEREBRUM AND CEREBELLUM—BASAL GANGLIA—CEREBELLUM —BLOOD-VESSELS OF THE BRAIN.

GENTLEMEN,—We must now return to some points that we passed over in considering the anatomy of the cerebrum. What is the course of the fibres that constitute the anterior limb of the internal capsule, lying between the body of the caudate nucleus and the lenticular nucleus? They pass, on the one hand, to the cortex of the prefrontal lobe, *i.e.*, the frontal lobe in front of the ascending frontal convolution. On the other hand, they descend into the crus, and occupy the inner (medial) portion of the crusta, lying to the medial side of the pyramidal tract. They descend to the pons, and there seem to end in the gray matter which is so abundantly scattered among the white fibres, longitudinal and transverse, of the anterior region of the pons. It is, however, probable that other fibres, proceeding from this gray matter to the cerebellum, continue the path to the cerebellar hemisphere, especially to the lateral and posterior regions. Thus this tract consists of fronto-cerebellar fibres. They degenerate downwards, and therefore probably conduct downwards; but this degeneration only extends to the pons, being arrested, as secondary degeneration always is, by the gray matter that interrupts their course. When the cerebellum is congenitally absent these fibres are also absent (Flechsig). It is probable that this connection is a crossed one, the frontal lobe on one side being connected with the cerebellar hemisphere of the opposite side.

I told you that the outer fibres of the crusta of the crus cerebri, the fibres that lie outside those of the pyramidal tract, do not pass up into the internal capsule, but radiate into the occipital and temporal lobes of the brain, passing from the crus, partly beneath the posterior extremity of the lenticular nucleus, partly between it and the external corpus geniculatum. These fibres connect these lobes with the cerebellum in the same way as the fibres of the inner part of the crus connect the frontal lobe with the cerebellum. Their connection is chiefly with the upper surface of the cerebellum, near the middle lobe, and it is probably also crossed. These fibres do not degenerate downwards, and therefore probably conduct upwards. They are also absent when there is no cerebellum.

The small bundles of fibres that lie behind, or rather above, the pyramidal tract in the crusta, between it and the substantia nigra (Fig. 5), consist of fibres that descend from the corpus striatum, caudate nucleus, and outer part of the lenticular nucleus, and, reaching the pons, probably connect those parts with the cerebellum in the same way as the other tracts connect with it the cortex.

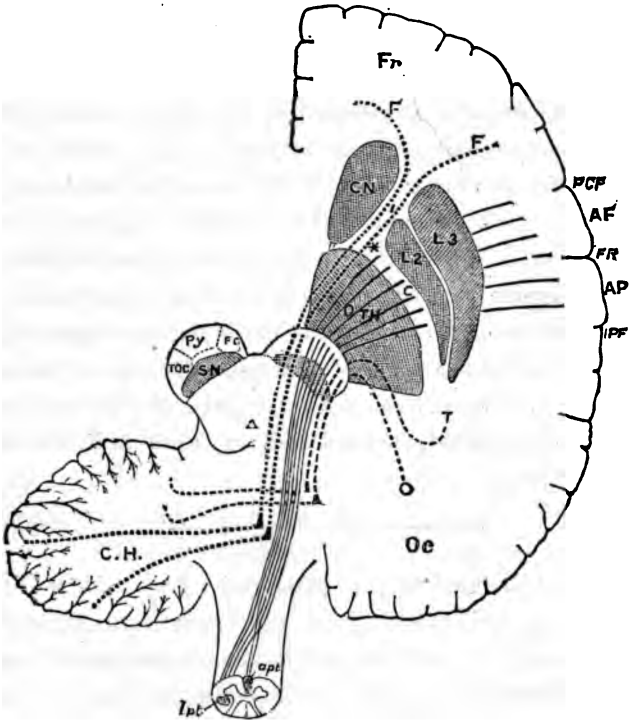

FIG. 12.—DIAGRAMMATIC HORIZONTAL SECTION, THROUGH THE CEREBRUM, PONS, AND OPPOSITE HEMISPHERE OF THE CEREBELLUM, TO SHOW THE COURSE OF THE FIBRES CONNECTING THE FRONTAL AND OCCIPITAL LOBES WITH THE CEREBELLUM.

In the section of the crus, on the left side, T O C shows the position of the fibres to the temporo-occipital lobe from the cerebellum (shown by dotted lines), and F C that of the fibres from the frontal lobe to the cerebellum (shown by broken lines) that occupy the anterior limb of the internal capsule. The other letters are explained on p. 10.

This extensive connection of the cortex with the hemispheres of the cerebellum is a fact of extreme interest, because the parts of the convolutions thus connected are those in which there is the least definite localization of motor and sensory function. In the prefrontal lobe of the human brain there is at present no evidence of motor function. In the temporal lobe we have only the auditory centre in the first convolution. In the occipital lobe we have only the visual centre, which, although not yet accurately defined, probably occupies only a part of it. In these portions of the brain a lesion may exist, and cause neither motor nor sensory symptoms; their function is more diffuse, so to speak, and thus capable of supplementary substitution. It is here that we must look for the processes concerned in the higher intellectual operations. These parts, whatever other connection they may have, seem to be connected with each other through the cerebellar hemisphere by the downward path from the frontal and the upward path to the temporal and occipital lobes. This very curious fact revives the old idea that the cerebellum is, in some way, concerned in intellectual processes—an idea suggested by the proportion that the cerebellar hemispheres bear to intellect as we ascend the scale of animals. Moreover, the cerebellar hemisphere has this in common with the parts of the cerebral cortex with which it is connected, that a lesion may exist in it without motor or sensory symptoms.

You may have observed with surprise that I have said nothing about the great central ganglia of the brain, which were formerly supposed to have such important connections with the motor and sensory tracts. It is probable that some of them have a connection with motor centres, but it is certain that their relations are very different in nature from those formerly attributed to them.

The optic thalamus receives fibres from below that come from the tegmentum of the crus, and probably these are derived from the upward sensory path from the spinal cord. It also receives fibres from the superior peduncle of the cerebellum; perhaps also from the optic nerves. From it fibres radiate to all parts of the cerebral cortex. Some go to the lenticular nucleus; but we do not know whether they end in it, or pass through it to the convolutions. Nevertheless, it does not seem to be in the path of those common sensations that affect consciousness. These pass, as we have seen, outside the hinder part of the thalamus, in the posterior third of the internal capsule. They are close to the thalamus, its lesions often involve them, but when they are unaffected there is no loss of sensation. It is highly probable, however, that the thalamus is concerned with some of the higher reflex processes.

The gray masses of the corpus striatum, on the other hand, seem to have no connection with the cortex. The caudate nucleus is connected with the cerebellum, as we have seen, by fibres that enter the internal capsule, either directly, or after passing through the lenticular nucleus, and lie in the crus close to the locus niger, reaching the cerebellum by the middle peduncles, after interruption by gray matter in the pons. No fibres from the caudate nucleus seem to join the pyramidal motor path. The lenticular nucleus, like the optic thalamus, receives fibres (chiefly collected into the "lenticular loop") from the tegmentum and from the opposite superior cerebellar peduncle. Thus both parts of the corpus striatum have an extensive connection with the cerebellum of the opposite side, and the descending fibres from the caudate nucleus degenerate (and therefore conduct) downwards, while those from the cerebellum to the lenticular nucleus probably conduct upwards. Thus this connection presents considerable analogy to that between the cortex and the cerebellum. In congenital absence of the cerebellum the corpus striatum is reduced to a third of its ordinary size (Flechsig). It seems probable that the two parts of the corpus striatum are central organs, analogous to the cortex itself. The analogy to those parts of the cortex that are connected with the cerebellum is rendered still greater by the fact that a lesion, even an extensive lesion, may exist in either the caudate or lenticular nucleus, and so long as it does not interfere with the functions of the motor or sensory parts of the internal capsule, it causes no symptoms.

Although the middle lobe of the cerebellum is continuous with the hemispheres, and the peculiar foliated cortex has a similar structure in both parts, it is certain that there is an essential difference in their function. In the white substance of the middle lobe are several curious nuclei of gray matter. As we descend the scale of animals the hemispheres become smaller, until the cerebellum, of birds, for instance, corresponds only to the middle lobe. The hemispheres may be diseased without recognizable symptoms, but this is not true of the middle lobe. The disease of this causes the peculiar unsteadiness of movement long known to be characteristic of cerebellar disease, and lately shown (by Nothnagel) to be characteristic only of disease of the middle lobe. The reason why the latency of lesions in the hemispheres was so long unrecognized is because the common lesion is tumor, and this almost always presses on the middle lobe. We must thus regard the middle lobe of the cerebellum as in some way concerned with the maintenance of equilibrium, perhaps by combining the afferent impressions, and arranging for harmonious centrifugal impulses. This view is supported not only by the effects of disease, but by the facts that certainly towards this lobe, and probably to it, fibres pass from the posterior columns of the cord, from the direct cerebellar tract (fibres from the lower trunk-muscles), and from the auditory nerve. Ferrier found that stimulation of the middle lobe caused movements of the eyes. Although we cannot conceive that centres for the voluntary movements of the eyes exist here, yet the eyeball muscles furnish important guidance to the centre that regulates the maintenance of equilibrium, and the centre for these muscles may thus be so related to the central lobe of the cerebellum, that its stimulation may cause indirectly the contraction of these muscles.

No part of the brain has excited more interest and received more study than the olivary bodies of the medulla; varied functions have been in turn ascribed them, but these hypotheses, destitute of foundation, have successively sunk out of view, and we are still ignorant of the function of these structures. Their connections are peculiar. The bodies resemble closely the dentate nuclei of the cerebellum; and the resemblance is certainly not accidental, for the two structures are connected by fibres, and atrophy together. Each olivary body receives fibres from the posterior columns of the cord on the opposite side, and is connected with the dentate nucleus, also of the opposite side. From each dentate nucleus fibres pass to the superior cerebellar peduncle and tegmentum of the opposite crus. Thus the tegmentum of one crus cerebri may be connected with the olivary body of the same side through the opposite dentate nucleus.

A large number of cerebral lesions are produced by disease of the cerebral vessels, by their occlusion or their rupture. To understand many facts about these lesions, you must know the arrangement of the cerebral vessels and the conditions of the cerebral circulation. I will briefly mention to you the facts of chief importance. Those that concern the arterial system have been ascertained by the investigations of Duret in France, and Heubner in Germany.

The blood-supply to the brain comes from the carotid and vertebral arteries. The left carotid arises from the aorta nearly in the direction of the current of blood that courses through the aortic arch; while the right carotid comes from the innominate, and this arises from the aorta nearly at right angles to the current of blood. Hence solid particles are rather more readily carried into the left than into the right carotid—a circumstance that explains the somewhat greater frequency of embolism on the left side of the brain. There is a similar but still greater difference in the origin of the two vertebrals, and it is probably for this reason that the left vertebral is often larger than the right. This does not determine any difference in the frequency of vascular lesions in the part of the cerebrum supplied, because the blood brought by both vertebrals has to pass through the common basilar. The internal carotid, on each side, divides into a small anterior cerebral, and larger middle cerebral. The latter continues the direction of the internal carotid, and hence plugs carried from the heart readily pass into it. The "circle of Willis," you will remember, is formed by these vessels, together with the anterior communicating artery, between the anterior cerebrals, and two posterior communicating (one on each side), between the middle and posterior cerebrals.

From the circle of Willis and the commencement of the three cerebral arteries (anterior, middle, and posterior) small branches arise which supply the central ganglia and adjacent white substance of the hemisphere, while the three arteries ramify over the surface of the brain, and supply the gray cortex and the chief part of the white substance. Thus there are two systems of branches, central and cortical. Between these two systems there are no anastomoses. The central branches do not communicate with each other, and hence obstruction of one causes necrosis of the region supplied, no collateral supply of blood being possible. The cortical branches vary in this respect in different individuals. In some persons there is enough communication between the cortical branches to maintain nutrition if one is obstructed; in other persons there is no communication. Hence obstruction of the middle cerebral at its origin always causes necrotic softening of the part of the central ganglia that it supplies, and sometimes also softening of the cortex; while in other cases, with a similar obstruction, we may find that the cortex is intact, although the central ganglia are extensively damaged.

The branches to the ganglia must engage our attention in further detail, since the pathological importance of these "central arteries" is very great. They are divided into six groups, of which two are medial and small, and four (two on each side) are lateral and very important. The anterior medial group is given off by the anterior cerebrals and anterior communicating artery, and supply the anterior extremity of the caudate nucleus. The posterior medial group consists of twigs given off by the posterior cerebrals near their origin. Passing through the posterior perforated spot, they supply the inner part of the optic thalamus and the walls of the third ventricle. These two medial groups are insignificant in the extent of the brain supplied by them, but of some pathological importance, since hæmorrhage, from their rupture, is apt to burst into the ventricles. The lateral groups furnish the blood to the chief part of the central ganglia and the internal capsule. The anterior lateral group consists of numerous small arteries that arise from the middle cerebral in the first inch of its course, pass through the anterior perforated space, and supply the caudate nucleus (except its head), the lenticular nucleus, internal capsule, and part of the optic thalamus. Some pass through the inner part of the lenticular nucleus to the internal capsule; while others pass first outside the lenticular nucleus, and then through its outer portion to the capsule. They supply the caudate nucleus and optic thalamus after passing through the capsule. These vessels are prone to rupture, perhaps because their direct origin from a comparatively large vessel exposes them to a high blood-pressure. Hence the frequency of cerebral hæmorrhage in this situation. The arteries of the posterior lateral group arise from the posterior cerebral, and supply the hinder part of the optic thalamus. Hæmorrhage from their rupture usually damages the posterior (sensory) part of the capsule, and often extends into the crus. Branches from the posterior cerebral supply also the crus and corpora quadrigemina.

Of the blood-supply to the cortex, that from the middle cerebral is both the most extensive and the most important, embracing, as it does, the central (motor) convolutions. The general arrangement of the branches of each artery is the same: each divides and ramifies, and from the branches, and the ultimate ramifications in the pia mater, twigs are given off to the cerebral substance—some short, that end in the gray cortex; others long, that pass through the gray cortex to the white substance, extending in it to various depths. The regions supplied by the several vessels are as follows:—The anterior cerebral, curving round the corpus callosum, supplies, by three branches, part of the orbital lobule, and the inner surface of the hemisphere as far as the quadrate lobule (precuneus). It also supplies, on the outer surface, by branches that come over from the inner surface, the first and second frontal convolutions, and the highest part of the ascending frontal. The middle cerebral, or "Sylvian artery," divides in the fissure of Sylvius, opposite the island of Reil, into four branches, which lie in the sulci of the insula, and then pass—the first to the inferior frontal convolution; the second to the ascending frontal, except the highest part, which is supplied by the anterior cerebral; the third to the whole of the ascending parietal, and the adjacent part of the inferior parietal lobule; the fourth to the convolutions about the posterior limb of the fissure of Sylvius, supramarginal and angular, the hinder part of the superior parietal lobule, and the first temporal. From this vessel, near its origin, one or two large branches arise that supply the greater part of the second and third temporal convolutions. The posterior cerebral supplies the occipital lobe, and also the inferior aspect of the temporal lobe, by three branches, of which one goes to the lower part of the uncinate convolution, a second to the inferior part of the temporal lobe, and a third to the cuneus, lingual convolution, and the inner and outer surface of the occipital lobe.

Thus the middle cerebral supplies the motor region, both central and cortical, except in part the leg-centre; it also supplies the part of the cortex that subserves cutaneous sensibility, the cortical auditory centre, and probably the higher visual centre; it supplies all the cortical regions concerned in speech-processes in the left hemisphere, motor, auditory, and visual. The anterior cerebral supplies only a small part of the motor region, viz., the part of the leg-centre that occupies the paracentral lobule and highest part of the ascending frontal. The posterior cerebral supplies the visual path, from the middle of the tract backwards, and the half-vision centre in the occipital lobe; it supplies also the corpora quadrigemina, and the sensory part of the internal capsule.

The pons and medulla receive small arteries from all the adjacent vessels, vertebrals, basilar, and cerebellar arteries. These branches are in two sets—median, near the middle line; and lateral or radicular, that pass in at the side, near the chief nerve-roots. Both pass back to the nuclei near the floor of the fourth ventricle, but the chief blood-supply to these nuclei comes from the median branches. Of the cerebellar arteries, the superior and inferior supply the corresponding regions of each hemisphere, and the upper surface is also supplied by a large branch from the basilar opposite the middle of the pons, the middle cerebellar artery. These arteries communicate freely, and hence necrotic softening of the cerebellum is rare. The branches to the medulla and pons do not communicate, and hence here necrotic softening is common.

The venous circulation of the brain presents several important peculiarities. The veins from the greater part of the cortex pass upwards to the longitudinal sinus, and open into this in a forward direction. This arrangement involves two very unusual conditions. Elsewhere, the blood from ascending arteries passes into descending veins, so that the feeble pressure that passes through the capillaries is supplemented by the influence of gravitation. Elsewhere, ascending veins convey blood that has been brought by descending arteries, and the venous flow is aided by the liquid pressure, which, according to the well-known law of hydrostatics, tends to make the blood rise in the veins. But on the brain, the blood from ascending arteries passes into ascending veins. The openings of these veins into the longitudinal sinus being directed forwards, the entering blood is opposed in direction to the current in the sinus, and the effect must be to retard the flow in both veins and sinus. Moreover, in the erect posture the anterior half of the longitudinal sinus has also an ascending course, while the trabeculæ that occupy the lumen of the sinus must offer some hindrance to the movement of the blood. These circumstances help us to understand the readiness with which clots form in the cortical veins and longitudinal sinus, when other circumstances favor the coagulation of the blood. Indeed, the marvel is that thrombosis is not more common than it is.

The veins of Galen, conveying blood from the ventricles to the straight sinus, pass above the corpora quadrigemina and middle lobe of the cerebellum, and are readily compressed by tumors in this situation. The course of the veins at the base is not of much medical importance. The blood from the internal ear passes into the cavernous sinus, that from the mastoid cells into the lateral sinus; and septic clots may thus extend when there is caries of the temporal bone. Many sinuses receive veins from the diploe of the skull.

There is very little communication between the individual veins of the surface, and hence obstruction of one causes grave damage to the cerebral tissue. The sinuses, however, communicate freely, and there are certain communications between the intracranial veins and those outside the skull. The veins of the nose communicate with the anterior extremity of the superior longitudinal sinus, and hence epistaxis relieves venous congestion within the skull. The ophthalmic vein (going to the cavernous sinus) communicates with the facial, and hence pressure on the sinus causes little or no distention of the retinal veins, because the pressure is quickly relieved. The mastoid veins effect a communication between the lateral sinus and the occipital veins. Moreover, many emissary veins pass through small foramina in the skull, and connect certain sinuses with external veins, the most important being between the longitudinal sinus and the veins of the scalp, and the inferior petrosal sinus and the deep veins in the neck. A further communication, variable in degree, is effected by the veins of the diploe. These communications are important, because they explain, first, the extension of morbid processes from the exterior to the interior of the skull, and secondly, the occasional occurrence of external tumefaction when intracranial sinuses are obstructed by thrombosis.

LECTURE IV.

SYMPTOMS OF BRAIN DISEASE: MECHANISM OF THEIR PRODUCTION—MOTOR PARALYSIS—HEMIPLEGIA.

GENTLEMEN,—You may remember that in describing to you the principles of the diagnosis of diseases of the spinal cord, I insisted on the importance of keeping not only distinct, but separate in your mind, the two parts of the diagnosis—the seat of the disease and the nature of the disease—the former indicated by the symptoms present, the latter by the mode in which they came on. The distinction is equally important in the diagnosis of diseases of the brain; but the separation is not equally practicable. The symptoms themselves are influenced by the nature of the disease to a far greater extent than in the case of diseases of the spinal cord. After we have ascertained the seat of the disease, we have to determine its nature, and then to consider how far the symptoms present are further explained by the character of the lesion. It is convenient to follow the method adopted in the case of diseases of the spinal cord, and, having considered in the last lecture the most important facts at present ascertained regarding the structure and functions of the brain, to study next the symptoms produced by its diseases, and afterwards the relation of the symptoms to the seat and nature of the morbid change.

Before we study the several symptoms, it is well to know the various mechanisms by which they are produced—mechanisms common to many morbid processes. The first of these is by the destruction of cerebral tissue. The function of the part destroyed is necessarily lost, and the loss is permanent unless it can be compensated by the action of some other part of the brain. In some parts of the brain the function is, as I have said, diffuse, and extensive compensation may occur. Although a loss of tissue, however small, doubtless has its effect, the evidence of the loss may be scarcely appreciable unless the lesion is extensive. When the loss does occur, it is manifested rather by a general lowering of function than by any special loss. This is the case with those regions of the brain that are probably concerned with the higher intellectual processes, as the prefrontal lobe. Some other functions are performed only by certain structures, and if these are destroyed, that function is permanently lost. Between these two groups there is another, in which special functions may not be permanently lost, even when the part of the brain subserving them is destroyed, because these functions are so related to both hemispheres that the corresponding part of the unaffected hemisphere can supplement that which is destroyed, and act for it. If, however, this second centre is also destroyed, the function is entirely and permanently lost.

Secondly, symptoms may depend on loss of function due to damage that falls short of destruction. The chief mechanisms of this damage are compression and defective supply of arterial blood. In compression both these mechanisms are combined; the pressure necessarily interferes with the flow of blood through the capillaries, and causes anæmia. Hence we do not know how far compresssion acts mechanically on the nerve-elements, and how far it acts by narrowing the vessels. A moderate compression of a nerve soon arrests conduction through it. If you compress your ulnar nerve behind the elbow, you soon cease to feel in the fingers supplied by it; but even here the compression must render the nerve anæmic, and we cannot infer that the effect on the fibres is simply mechanical. In the brain there is a curious fact regarding pressure which you should remember. Pressure is very much more effective when it is suddenly produced than when it is slowly produced. The pressure of a cerebral hemorrhage causes symptoms (that we can certainly refer to the pressure) of much greater intensity than does a tumor, although the latter may be of larger size. It is easier to explain this difference if we assume that pressure acts mechanically than if we suppose that it only causes symptoms by producing anæmia.

A diminished supply of arterial blood also causes loss of function. If the supply is altogether cut off, the loss of function is immediate and absolute. This is true of nerve-fibres as well as of nerve-cells. For a short time, perhaps a day, function may return if the blood-supply is restored; afterwards structural disintegration occurs, and the nerve-elements perish. Remember that arterial blood may be deficient when there is no absolute diminution in the amount of blood in the part. If there is a hindrance to the return of blood by the veins, the over-filled vessels cannot receive blood from the arteries, and so the symptoms of mechanical congestion are to a large extent the same as those of anæmia.

The nerve-elements may be damaged or destroyed by more minute morbid processes, either beginning in the interstitial tissue or in the nerve-elements themselves, such as the various processes of inflammation and degeneration.

The second disturbance of function that results from brain-disease is that which we call "irritation." Irritation causes two effects. First, there may be a morbid increase of activity instead of a diminution; there is evidence of an excessive, although abnormal, liberation of energy. This disturbance may be sudden and paroxysmal, or persistent. The former is often spoken of as "discharge," by an obvious metaphor, which is, indeed, more than a metaphor. Discharge implies a preceding charge. The nerve-energy liberated in the discharge must have been ready for liberation, but restrained. It must have been a state of "tension"—"held," that is to say. But "held" by what resistance? We do not know. Nevertheless, the fact of a resistance and restraint, co-extensive with the production of nerve-force, helps us much in understanding the phenomena of nerve-action in both health and disease. It helps us, for instance, to comprehend what has seemed to some so strange a paradox, the fact that disease should cause over-activity. If we conceive, as by all analogy we may, that the restraint is the highest function of nerve-cells,—that the self-control, and the capacity for being controlled, are higher functions than liberation of energy,—we can understand that when, by disease, there is deterioration of function, one effect of this is excessive activity. When the brain is suddenly deprived of blood, one effect often is to cause convulsions: thus the first result of failing function may be the liberation of energy. No doubt in irritation the same process is operative. At the same time we must not deny that some influences may directly augment the energy-producing action of cells, although we have no means of proving such an augmentation.

These considerations enable us to understand something of another and remarkable fact—the second effect of irritation. It may not only cause over-activity of nerve-elements, it may lessen their activity, and even arrest it. This arrest is an example of what physiologists term "inhibition." It may be conceived as an increase of the resistance or restraint. It is remarkable that the same process should sometimes prevent and sometimes permit the liberation of nerve-force, but instances of this are familiar to physiologists. The same stimulus, in different degrees, will either arrest or produce reflex action. In irritation the nerve-tissues directly affected may be inhibited or discharged, or their irritation may inhibit or discharge connected nerve-cells at a distance. It is probable that the nervous system is full of mechanisms whereby the action of certain centres is controlled by that of other centres, and it is probable that the chief mechanism of this association is control, and that what we call the excitation of one centre by another may be very often simply a lowering of control, permitting activity. Thus we can understand that inhibition, as well as excitation, may be a result of the pathological process that we call irritation.

Almost all organic lesions of the brain involve these two processes—damage, complete or incomplete, and irritation. Their relative degree varies, and still more does their relative duration. In a sudden lesion there is immediate damage and immediate irritation. The irritation soon passes off, unless it is maintained by a secondary more chronic process. The damage that is incomplete also passes away, and with it the symptoms that it has caused. The damage that is complete persists, and its symptoms persist. Hence the symptoms of an acute lesion of the brain are at first far wider and more severe than correspond to the actual destruction. The excess due to slighter damage (as by compression) and to irritation, soon passes away. It has become customary (in Germany especially) to distinguish the two classes as the "direct" and "indirect" symptoms. The terms are convenient, although they are not exact, since almost all the so-called indirect symptoms are, in one sense, the direct effect of the lesion.

In disease that is gradual in development and course, such disease as a tumor, slighter damage and irritation are constantly occurring. The symptoms due to these accompany those due to the destruction by the disease. Hence the symptoms of such disease are often complex, and far more extensive than might have been anticipated. You may now see the meaning of the statement I made just now, that the nature of the lesion has far more influence on the character of the symptoms in disease of the brain, than it has in disease of the spinal cord.

The division into direct and indirect symptoms is founded on the mechanism by which they are produced, and the distinction, as we have seen, is not merely theoretical, but is based on the important fact that, in the case of acute lesions, the indirect symptoms, however obtrusive at the onset, soon pass away, while the direct symptoms persist. Unless the loss can be compensated, it persists as long as life endures. We have now to consider another important division of the symptoms, founded, not on their mechanism, but on their character. Some symptoms, such as local palsy, are due to, and indicate, interference with the function of a definite part of the brain. These are termed "*focal*" symptoms, because they are due to disease at a given spot. It is not quite in harmony with the modern sense of "focus," although there is always a tendency to associate concentration with limitation; but the sense is not altogether alien to the original meaning of "focus," which, you will remember, is that of a fireplace. Other symptoms, such as loss of consciousness, or delirium, indicate a widespread interference with the function of the brain, and are called "*diffuse.*" This distinction, although important and useful, must not be conceived as absolute. Few of our distinctions are absolute. Some symptoms may be diffuse in one case, focal in another, as, for instance, convulsions. Moreover, diffuse processes may cause focal symptoms, and *vice versâ*. Both direct and indirect symptoms may be either focal or diffuse; but it is much more common for direct symptoms to be focal than diffuse, and somewhat more common for indirect symptoms to be diffuse than focal.

In considering in detail the symptoms, irrespective of their cause, we will not follow a strictly logical order, but will first take the symptom that is one of the most common, which we know most about, and therefore should be able to understand best—loss of the power of voluntary motion, motor paralysis. The loss of power may be complete or partial in degree; both have always been, and still are, commonly termed "paralyses." It has of late become fashionable to call the partial loss "paresis," a term of doubtful value except as a means of giving a questionable satisfaction to patients, who find comfort in the mysterious word, and think well of its donor—at any rate until the next physician whom they consult assures them that the disease *is* "paralysis," and that "paresis" is only Greek for weakness. But what is altogether unjustifiable, is to assert that partial loss of power is *not* paralysis.

Impairment of motor power is due to interference with the motor centres in the cortex of the brain, or the motor path from them by the internal capsule, crus, pons, and pyramids of the medulla, in the course that we have already traced. Above the pons the two paths are separate, and a lesion in one crus, or one hemisphere, affects only one motor tract, causing paralysis of the opposite half of the body—"hemiplegia." In the pons the two paths are near together; both may be affected by a single lesion, and yet they are far enough apart for one to be often affected alone. In the anterior pyramids of the medulla they are so near that both often suffer. A lesion here, on one side, affects the arm and leg on the opposite side. Above the medulla the path from the hypoglossal nucleus, having crossed the middle line just above the nucleus, is associated with the tract for the limbs, and so the tongue is paralyzed on the same side as the limbs. Above the middle of the pons the facial path joins that of the limbs, and the hemiplegia involves the face. Thus affection of face, tongue, arm, and leg on the same side is the characteristic of complete hemiplegia that results from disease anywhere between the cortex and the middle of the pons. The fibres of the motor path spread out in passing from the crus into the internal capsule, and still more widely in passing through the white substance to the cortex (see Fig. 5, p. 10), the tracts for tongue, face, arm, and leg being to a considerable extent separate, and the separation being greatest at the cortex. Hence, a lesion, even of some size, in the cortex, or in the white substance beneath the cortex, may affect only one or two of these parts, the other escaping. The arm, for instance, is often thus paralyzed alone. But even a small lesion of the internal capsule usually affects all the tracts; it must be very small indeed to damage only one of them. If the lesion is in the crus, although it is very small, all are involved. Theoretically, it is true, a lesion in the crus or pons may be so small as to damage only one part; but practically this is scarcely ever met with.

The hemiplegia is always on the side opposite to the cerebral lesion. A few cases have been recorded in which the hemiplegia was on the same side as the lesion, and these have exercised very much the minds of pathologists. But a much more frequent event is to meet with hemiplegia without any discoverable lesion in either hemisphere. Some morbid change must exist in such cases; and a similar undiscovered lesion in the hemisphere opposite to the paralysis is the most probable explanation of the cases in which the only discovered lesion is on the same side as the hemiplegia. Coexistence does not necessarily involve causation.[1]

In a case of severe hemiplegia—"complete hemiplegia," as it is called—the paralysis affects one side; but not the whole of one side. The arm and leg are powerless; the face is paralyzed chiefly in the lower part; the upper part of the face moves almost as well as on the unparalyzed side. The tongue, when protruded, deviates towards the paralyzed side (being pushed over by the opposite unopposed genio-glossus); but the muscles of mastication contract equally, or almost equally, in ordinary action, and the two sides of the thorax move equally in ordinary breathing, or if there is an inequality it passes away in a few days. But if the patient makes a strong effort, the masseter on the paralyzed side does not contract quite so strongly as the other, and if he takes a deep breath, and brings into action the extraordinary muscles of respiration, there is a distinct defect of expansion of the corresponding half of the thorax. I remember when I was a student, learning from two distinguished physicians—from one that the masseters and respiratory muscles are always weakened in hemiplegia; from the other, that they never are. Both were right, or nearly right. The one had observed only ordinary movements, the other extraordinary movements. Other muscles of the trunk are also weakened—those, for instance, of the back and of the abdomen,—but the degree of weakness is always slight.

Thus some muscles are completely paralyzed, some are merely weakened, others are usually not paralyzed at all, and are never paralyzed much. Moreover, this does not depend on the extent of the disease in the brain. It occurs when the whole of one motor tract is destroyed. This is best explained by an hypothesis, first suggested (in a slightly different form) by Broadbent. Some muscles are habitually used without their fellows on the other side—as the muscles of one arm. Others are often used with their fellows, but often also alone, as the muscles of extraordinary respiration. Others are never used without their fellows, as the intercostals, the frontales, and the masseters. The degree of paralysis in hemiplegia corresponds roughly to the degree of unilateral use. The muscles of bilateral use are represented in both hemispheres of the brain, and the degree of bilateral representation corresponds to the degree of bilateral use. It may be that the representation of these muscles is rather greater in the opposite hemisphere than in the hemisphere of the same side, or that the nervous arrangements are in greater functional activity in the opposite hemisphere. Hence there is sometimes slight weakness for a short time after the onset of hemiplegia; but the hemisphere of the same side is soon able to innervate them in full degree. It is possible, although not yet proved, that the innervation is affected by pyramidal fibres that do not decussate.

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[1] The hypothesis of Morgagni, that there is no decussation on the medulla in these cases, does not receive so much support as might seem from the discovery of the variability of this decussation, because it is certain that the medullary decussation, when deficient, is supplemented in the spinal cord.

I have said that though muscles of bilateral use may be at first weakened, the weakness soon passes off. In muscles that are of partial bilateral use the weakness also lessens, although less quickly. The legs are used much together, although capable of unilateral use, and the loss of power in the leg never remains absolute, although it may remain absolute in the arm. As a rule, however extensive the lesion, the patient regains ultimately some power in the leg, and commonly enough power to enable him to stand. When hemiplegia occurs in early life, the leg always recovers, and becomes as strong as the other, even when its growth is retarded. Since this occurs when the lesion involves the whole of the motor tract of one hemisphere, it must be through the unaffected hemisphere gaining by use that power over the leg of the same side for which, doubtless, structural arrangements always exist, although they are not called into complete functional activity under normal conditions.

Another effect follows from this double representation of the leg. The lesion of the brain causes at first some weakness of the leg on the same side as well as on the other, but this soon passes off. Doubtless, if we could measure their strength, the other bilateral muscles would exhibit the same bilateral weakness.

These phenomena present also another aspect. Most of these bilateral movements are automatic—need little or no voluntary effort. Movements are lost in proportion as they require will; persist in proportion as they are automatic. Again, emotional movements are automatic: the will is needed not to cause, but to restrain them. Emotional movement may be preserved when voluntary movement of the same muscle is lost. For instance, if the patient tries to show his teeth, the mouth may be motionless on the paralyzed side, and yet if he smiles there may be little or no difference between the two sides. Emotional movements are probably innervated from either hemisphere.

Certain movements are normally effected by non-corresponding muscles of the two sides. Such, for instance, are the lateral movement of the eyes, and the rotation of the head. In the latter the head is turned to one side by some muscles of that side acting with the sterno-mastoid of the other. In hemiplegia these movements are affected, but chiefly during the early stage. It is the movement towards the paralyzed side that is lost, and the unopposed antagonists may even cause a slight deviation of the head and eyes towards the unaffected side, *i.e.*, towards the side of the brain diseased—this is termed "conjugate deviation." It shows us very clearly that movements rather than mucles are represented in the brain. The fact that the conjugate deviation occurs, and that it passes away, shows us two things. It proves, first, that these movements, though effected by muscles of both sides, are habitually innervated from the opposite hemisphere, *i.e.*, the head is turned to the right by the left hemisphere of the brain. Secondly, it shows that the movements are also represented in the hemisphere of the same side, by nervous arrangements that may readily be called into effective use.

If a patient is unconscious, we can no longer call his will into action, and are thus deprived of the direct evidence of loss of voluntary power. If the patient is restless, the absence of movement on one side may be observed; and a pinch of the skin may cause a movement on one side only. There may also be flaccidity of the muscles of the paralyzed side, and the limbs, when raised, fall more suddenly than on the unaffected side; or, on the other hand, they may present a distinctly abnormal rigidity. The conjugate deviation of the head and eyes, if present, also indicates a one-sided loss. Moreover, we may sometimes obtain help by observing the state of reflex action.

It is very common, although not invariable, for the reflex action from the skin to be lessened or abolished on the paralyzed side. The loss may be observed in the plantar, cremasteric, and abdominal reflexes. It exists from the onset. Why this symptom exists in some cases and not in all, we do not yet know.

The myotatic irritability, evidenced by the so-called "tendon-reflexes," is often unchanged during the first week, and at the end of that time becomes excessive, so that the foot-clonus can be obtained. Sometimes, however, a clonus can be obtained a day or two after the onset, and there may be, immediately after the onset, complete loss of these reactions, so that even the knee-jerk cannot be obtained. The early change is to be ascribed to an influence exerted by the lesion on the spinal centres; the later increase to the secondary degenerative changes in the pyramidal tracts reaching these centres.[1]

At some period the muscles of the paralyzed limbs become rigid, stiffening the limbs in certain postures, and opposing passive movement. Todd first distinguished between "early" and "late" rigidity. The former comes on a few days after the onset, and lasts for a few weeks. The posture of the limbs is that of rest. It is probably due to the irritation of the fibres by inflammatory changes about the lesion. But there is sometimes an "initial rigidity," which develops immediately, and lasts for a few hours or for a day or two. It is probably due to the irritation of the fibres by the lesion itself. When these forms of rigidity are considerable in degree we can often obtain the foot-clonus. The rigidity is the result of that state of the muscle-reflex centres in which myotatic irritability is increased. Late rigidity comes on in the course of a few weeks, and persists as long as the palsy. The shoulder is adducted, the elbow flexed, the wrist pronated and slightly flexed; the fingers are strongly flexed at the middle and distal phalangeal joints by the contracture of the long flexor. When the wrist is passively flexed, so as to shorten the course of the flexor tendons, the fingers can be straightened without difficulty. Although the flexor contracture preponderates, the extensors also present some rigidity. In the leg the rigidity is more nearly equal in the two sets of muscles, so as to fix the limb in the position of extension. This rigidity depends on active muscular contracture. It lessens much during sleep and when the limb is warm. It can be overcome, for the time, by gentle prolonged extension, especially if the muscles are simultaneously rubbed. After some years tissue-changes take place in the muscles, and they can no longer be extended. Thus we ought to distinguish from the late rigidity an ultimate structural contracture, making in all four varieties—initial, early, late, and structural rigidity. The late rigidity coincides with degeneration in the pyramidal tracts of the cord. Excess of myotatic irritability accompanies it, as it does the degeneration of spinal origin, no doubt for the same reason. The foot-clonus and rectus-clonus can readily be obtained; in the arm, a tap on a muscle, its tendon, or the bone to which it is attached, causes a momentary contraction, and sudden tension may develop a clonus in the flexors of the fingers, and sometimes in the flexors of the elbow, and even in the trapezius.

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[1] See "Diagnosis of Diseases of the Spinal Cord," 3d Ed., p. 23, for a fuller discussion of this subject.

LECTURE V.

SYMPTOMS (*Continued*): HEMIPLEGIA (*Continued*)—CONVULSION.

GENTLEMEN,—We considered, in the last lecture, the chief characters of hemiplegia, and the condition of the muscles in lasting palsy. Some other points regarding the state of the limbs remain for consideration, the first being the changes that may occur in their nutrition.

The nutrition of the muscles may be unchanged, even after the paralysis has existed for years, or slight general wasting may set in a few weeks after the onset, sometimes slowly attaining a considerable degree, although never comparable to that in progressive muscular atrophy. The electric irritability of the muscles may present no change, or a slight increase in irritability may occur at the end of one or two weeks, and continue for a few months, to give place to a slight and permanent diminution. The change is the same to both forms of electricity, faradaism and voltaism, and is the same in the nerve-trunks as in the muscles. It occurs chiefly when there is the change in nutrition just described, and each is probably the consequence of the irritative character of both the cerebral lesion and the resulting secondary degeneration of the pyramidal tract in the cord. Although this degeneration never invades the motor nerve-cells as a destructive change, it seems to influence, in slight degree, their nutrition, and therefore that of the motor nerve-fibres and muscles.

Vaso-motor and trophic changes may be absent or very marked. They appear to depend in part, like the changes in muscular nutrition, on the irritative character of the cerebral lesion; but there are centres in the cortex that influence the vaso-motor state of the limbs, and disease of these centres, or of the downward path from them, the precise position and course of which are still undetermined, may be the cause of considerable disturbance of this character. During the early weeks there is often increased warmth of the paralyzed limbs, amounting to from half a degree to a degree and a half Fahrenheit, at first uniform, afterwards intermitting. With this there may be increased redness and lividity, sometimes with marked œdema, especially if the kidneys are also diseased. Often there is a tendency to graver trophic changes; blisters readily form, filled with dark serum; the skin sloughs from slight pressure in those parts on which pressure chiefly acts in the recumbent posture—the gluteal region, over the trochanter and malleolus. Rarely there is inflammation of joints.

When recovery occurs, power returns in the proximal parts of the limbs sooner than in the distal parts, and in the leg before the arm. Indeed, as already stated, some recovery in the leg is invariable. There is, moreover, more use of the leg in association with the other, than in its separate movements. The flexion of the foot is that which remains longest defective, and hence the patient cannot get the toes off the ground in bringing the foot forward in the act of walking, and swings the leg round. In the arm, the shoulder recovers before the elbow, and the elbow before the hand. The extensors remain weak longer than the flexors, and the supinators longer than the pronators. In rare cases the hand-movements return first; and it is singular that these cases sometimes also present another exceptional feature—the arm improves faster than the leg. No doubt this peculiarity depends on a special position of the lesion.

The distribution of the palsy in cases of hemiplegia depends on the position and extent of the lesion, and certain forms need special notice. I have already mentioned the escape of the tongue on the affected side, when the lesion is in the medulla, and of the face when it is in the lower half of the pons; that is to say, when the lesion occurs before the cerebral path from the hypoglossal and facial nucleus has crossed the middle line, and has become associated with the path to the limbs. But the cranial *nerves*, from the third to the hypoglossal, in passing from their nuclei to the surface, may be damaged by disease that damages also the motor tract. This paralyzes the nerve on the same side as the lesion, but the limbs on the opposite side to the lesion, thus causing what has been badly termed "alternate hemiplegia." Certain nerves are more frequently paralyzed in this way than others; the most frequent are the facial, the sixth, and third nerves; less frequently the hypoglossal, auditory, and fifth. The facial and sixth are sometimes affected together on the side opposite to the limbs. The paralysis of the face resembles that due to other diseases of its nerve; all parts of the face are paralyzed, and there is loss of faradic and preservation of voltaic irritability. When the lesion is in the crus, the face is affected on the same side as the limbs, but the third nerve on the opposite side—on the same side as the lesion. Do not imagine that this crossed palsy of limbs and cranial nerves is invariable when hemiplegia results from disease in these regions. The lesion may be so placed, or so small, that the nerves escape. Thus the association of palsy of cranial nerves on one side, with that of the limbs on the other, gives us one class of varieties of hemiplegia.

Another class depends on the incomplete extent of the palsy, the seat of the disease being in the cerebral hemisphere, where the constituent elements of the motor tract have so far diverged that its damage may easily be partial. We have already seen that this must be rare in lesions of the internal capsule, on account of the proximity of the several paths, and that it may more readily occur in the white substance, and most readily in or beneath the convolutions. The paralysis may involve only the face, or the arm, or the leg: or it may involve the face and tongue; face, tongue, and arm; or face and arm. The distribution depends on the relative position of the centres and paths. Thus the tongue and arm are never affected by a single lesion without the face, because the face-centre and path intervene between the two others. Similarly, the face and leg are never affected without the arm, because the centre and path for the arm intervene between the others. This partial hemiplegia is sometimes called "monoplegia," distinguished, according to its seat, as lingual, facial, brachial, or crural, while combinations receive compound names, as brachio-facial monoplegia. It may seem to you rather anomalous to call the latter a monoplegia, but the whole nomenclature is inconsistent. Strictly, "monoplegia" should designate double hemiplegia; but we call this "diplegia." Thus two "half-palsies" make, not a "one-palsy," but a "two-palsy," and a "one-palsy" is less than a "half-palsy."

In these cases of partial hemiplegia, the paralysis never remains absolute, and usually is not absolute, even at first. The state of the limb resembles that of a hemiplegia that is recovering. The coarse movements in the upper part of the limb are preserved, while the movements of the extremity are impaired or lost.

The affection of sensation that often accompanies hemiplegia I shall describe presently. Before leaving the subject of motor palsy one curious class of symptoms must be mentioned. These are the disorders of movement that sometimes come on some months after the onset. The rigidity of which I have already spoken is fixed, varying but little during the waking hours. But the muscular contractions we are now considering are versatile and changing. They also vary much in different cases. Sometimes there is tremor; fine, quick, rhythmical contractions of the muscles. Rarely there are slow and rhythmical movements, wider in range, and chiefly met with in the hands or fingers. Most common of all are irregular muscular contractions, irregular both in time and in degree, rarely quick, far more often slow. The quick movements somewhat resemble those of chorea, and hence the term, "post-hemiplegic chorea," has been applied to the whole class of movements—unwisely, because they have nothing to do with chorea, and; moreover, resemble chorea only in rare cases. Generally, the movements are far slower than are seen in true chorea. If not constant, they are readily evoked by an attempt at voluntary movement, or even by attention, and by these they are always increased. Inco-ordination of movement results, peculiar in character—slow, irregular, spreading movements of the fingers, that have been compared, not inaptly, to the movements of the arms of a cuttle-fish. "Mobile spasm," it may be conveniently termed. With this there is often some more constant and unchanging spasm, especially in the flexors of the wrist. The arm is always involved in greater degree than the leg. In the latter the effect of the spasm is chiefly to cause inversion of the foot and over-extension of the great toe; spontaneous movements are rare. The arm is usually adducted at the shoulder-joint; the elbow is sometimes flexed, sometimes strongly extended. Often the arm, straightened out, is carried behind the body. The wrist is frequently flexed; the fingers are usually flexed at the metacarpo-phalangeal joints, extended, and even over-extended at the others, the spasm preponderating in the interosseal muscles. There may be a subluxation of the extended finger-joints, the heads of the phalanges projecting on the palmar aspect. Thus there is a remarkable contrast between this spasm and ordinary late rigidity. In the latter the spasm chiefly affects the long flexors of the fingers; the digits are bent at all joints, a form of flexion that is employed in coarse movements of the limb. In the mobile spasm there is the "interosseal flexion" just described, the flexion that is employed in many delicate operations, such as the act of writing. The continuous action of the muscles often leads to their overgrowth, and the limb may be actually larger in circumference than that of the opposite side, when it is less in length. This condition sometimes develops without preceding hemiplegia; and to such a case, in which there was no fixed spasm, but only the slowly-changing irregular movements, Hammond (of New York) gave the name of "athetosis" (= without fixed position). In the vast majority of cases the condition is a sequel to hemiplegia. It sometimes comes on after hemiplegia in adult life, but is far more frequent after hemiplegia in infancy and childhood, to which it is, indeed, the common sequel. In adults it has been observed chiefly in cases in which the lesion was in or near the optic thalamus. Why, we cannot yet say. In children it does not seem to be related to any special seat; it follows disease anywhere in the motor regions of the hemisphere. The lesion causing the initial hemiplegia is, however, almost invariably softening, and not hæmorrhage. An analysis of adult cases shows this clearly; and in children any other acute lesion than softening is very rare. These considerations suggest that one element in its causation may be the partial recovery of nerve-cells that are damaged, but not destroyed—which recover, but with disordered functions,—and the greater power of recovery and greater capacity for derangement during the period of development may be the cause of the special frequency of this condition after hemiplegia in early life.

From motor palsy, the spasm last considered naturally leads us to that paroxysmal over-action which causes convulsion. Convulsions are frequent and important symptoms of cerebral disease. They occur under two conditions, apparently as the result of two different mechanisms. First, they occur if there is active irritation of the brain-tissue, such as is produced by inflammation of the brain or membranes, a growing tumor, or a sudden lesion. Secondly, they occur in what are termed "stationary lesions," in which the stage of activity is over, and such structural recovery as may be possible is taking place, or has taken place. In this case they are, apparently, due to the imperfect recovery of damaged nerve-cells, which regain the power of evolving nerve-force, but not the higher power of regulating its discharge. By each mechanism, convulsions are produced most readily when the disease is in the cortex. Stationary lesions scarcely ever cause convulsions unless they are situated in or near the motor cortex. Active irritation is most effective when in the same region, but it may cause convulsions whatever be its seat. They are also produced by general increase of intracranial pressure, and by diffuse processes, such as meningitis.

The diagnostic significance of convulsions depends on their character. They may be general or they may be partial, either in extent or in commencement. General convulsions constitute a "diffuse" symptom. They are often due to a diffuse and widespread morbid process, inflammatory or degenerative disease away from the motor centres, or to general increase of intracranial pressure. Convulsions that are limited in extent or commencement constitute a focal symptom, and indicate disease in or near the motor region, especially the motor region of the cortex. In general convulsions the loss of consciousness is sudden and immediate; there is usually no aura. In partial convulsions, consciousness is lost late, and the patient is usually aware of the local onset. This local onset is due to the fact that the discharge begins in the centre irritated—for the face, arm, or leg. If very slight, it may not spread beyond the one centre in which it began, the convulsion being confined to the corresponding part. If more severe, it spreads to all the centres of that hemisphere, and the convulsion affects the whole of one side of the body. If still more severe, the discharge spreads to the other hemisphere, and the limbs of the other side are also involved, usually after the first side, sometimes, in the most severe fits, simultaneously. Consciousness is usually retained throughout when the convulsion is confined to one limb; sometimes when it involves the whole of one side; scarcely ever when both sides are involved. Different discharges vary in intensity, and the patient often has slight attacks that are local, and more severe attacks that begin locally and become general. The attack usually commences by clonic spasm, which often becomes tonic if the convulsion becomes severe. The spasm almost always begins in the extremity of the limb. But it is common for a sensory "aura" in the part to precede the spasm; the sensation may pass up the limb first affected, along the side of the trunk, and down the second limb affected, and only then may spasm be added. It is as if the discharge commenced in the sensory cells of the cortex, passed like a ripple through these sensory centres, and only when it reached their limit was deflected to the motor cells. In the very slightest attacks the sensory aura may be accompanied, not by spasm, but by the opposite condition—sudden powerlessness of the limb. The influence of the sensory discharge on the motor cells may thus be to inhibit them, if it is of very slight degree—another instance of the relation of inhibition and discharge mentioned on p. 40. Special sense auræ are very rare in organic disease; when they occur they indicate that the disease is in or near the part of the cortex that subserves the corresponding functions. Thus, I have met with a case in which a flash of light was the aura of fits due to a tumor of the occipital lobe; and another, of a tumor beneath the temporo-sphenoidal cortex, in which the sound of bells was the aura.

A convulsion leaves behind it a transient weakness in the part convulsed, probably due to exhaustion when the spasm is severe, or to motor inhibition when the spasm is slight or the discharge is limited to the sensory cells, sometimes perhaps to both mechanisms. The weakness after a unilateral fit may resemble slight hemiplegia, and, if the convulsions recur at short intervals, the residual palsy accumulates until it may amount to absolute powerlessness of the side, with increased knee-jerk and foot-clonus. It gradually passes away after the convulsions are over.

The characteristic, then, of the convulsions of organic brain-disease is their local commencement. Whether the fit remains limited to the part in which it commences, or spreads to other parts, even the whole body, is a question of degree. Moreover, a local aura, without convulsion, has the same significance as local spasm. It indicates that the discharge commences at one part of the brain, that there is at that part a morbid state, and it is thus a focal symptom. Convulsions that are general from the first are often due to, and symptoms of, organic brain-disease; but they derive their significance from associated symptoms, and not from their character. Without such symptoms they suggest idiopathic epilepsy, not the organic brain-disease that local commencement suggests. I say *suggests,* because there is sometimes a local commencement in idiopathic epilepsy. This is, however, rare, and you would not think of idiopathic epilepsy in such a case unless you had ascertained that other symptoms of organic brain-disease were absent, and you could not feel confident, unless the convulsions had existed alone for a considerable time, or there was a strong family tendency to the idiopathic disease.

The first convulsions from organic disease are sometimes excited by a cause outside the central nervous system, a cause that may seem to be adequate to account for them. This is a very important point. Remember that the apparent cause of a fit is scarcely ever more than its excitant—an excitant that is effective only because there is a predisposition; and the predisposition is the important matter to be sought out and to be treated. The spark would be harmless unless it fell on gunpowder. The predisposition may be sometimes an inherited tendency of the nervous system, or it may be an acquired state of morbid irritability, such as forms part of the constitutional disease we call "rickets," or it may be a brain disease that has not reached such a degree as to cause pronounced symptoms. For instance, a child was brought in here who had swallowed a slate pencil, and then had an attack of general convulsions. For this the slate pencil seemed an adequate cause. It was passed by the bowel, and the child seemed well. But six weeks afterwards she was dead, and the cause of death was a large infiltrating glioma of the pons, which must have existed at the time of the convulsion, and no doubt gave rise to the predisposition. As in this case, the convulsion so excited is usually general. But only a few weeks ago, you may remember, we had a man in Ward IV., whose history showed that a convulsion beginning locally may be excited by a local cause, even though it ultimately depends on organic brain-disease. The man, when apparently well, struck his left arm against an iron hook, and a few hours afterwards had a fit which began in this arm, and was heralded by a pain at the part struck. He afterwards had other similar fits. It seemed like a case of what is termed "reflex epilepsy"—a disease, by the way, much more rare than you may imagine from books. But one day he had a fit which began in the foot, and not in the arm. Then he developed headache, double optic neuritis, and hemiplegia; and when he came in here, he presented the characteristic symptoms of a tumor in the upper part of the ascending frontal convolution. The blow on the arm had determined the time and place of an explosion, of which the real cause was the organic brain-disease. Such a case is the converse of the arrest of a fit, beginning locally, by a ligature round the limb, when the fit is due to organic brain-disease, and the ligature can only stop the fit by acting on the centre in the brain.

Remember, then, that convulsions should not be lightly dismissed from consideration because the fit succeeded an exciting cause.

Sufferers from organic brain-disease who are of the age and sex in which the predisposition to hysteria exists, may suffer from attacks of hysteroid convulsion. It is not surprising that so potent a disturbing influence should evoke into activity a tendency from which few women are altogether free. But the fact is of extreme diagnostic importance, because, when one of a set of symptoms is unmistakable in character, it is natural to take it as an index to the series. You should never do so, gentlemen, until you have satisfied yourselves that no symptom is certainly of a different nature. I insisted on this in speaking of the diagnosis of diseases of the spinal cord, and it is equally true of those of the brain. Both acute and chronic disease may lead to the strange disturbance of function to which we give the name hysteria, and which we might just as well call, as did our forefathers, "fits of the mother." I have seen it, for instance, many times in cases of cerebral tumor, many times in tubercular meningitis, and once at the onset of an attack of embolic hemiplegia. It is a very frequent consequence of the defective development of the brain that results from infantile lesions. Some of you may remember a curious girl who attended here for a long time, whose powerless and withered arm was the relic of a severe infantile hemiplegia. She suffered from unilateral epileptiform convulsions, and also from pure hysteroid fits of intense severity, as well as from laryngeal spasm, phantom tumor, and various other symptoms of the same class.

Tonic spasm, in the form of muscular rigidity, occurs as a chronic symptom chiefly after hemiplegia, and as an acute and subacute symptom in irritating lesions, especially in inflammation of brain and membranes, either primary, or secondary to necrotic softening. It is almost always one-sided. Severe tetaniform spasm is rare. It is generally paroxysmal, and has been met with chiefly in disease of the cerebellum, but only in cases of tumor, and it is probably due, not to the damage to the cerebellum, but to the pressure of the tumor on the pons. Similar spasm may be caused by tumors of the pons. Forced movements, as a tendency to rotation, are extremely rare. They resemble those produced in animals by injury to the semicircular canals or cerebellum, and have been observed chiefly when disease involved the middle cerebellar peduncle. Of their precise mechanism we are still ignorant. Fine tremor is rare except after hemiplegia, but sometimes attends muscular weakness in chronic disease. It is without special significance. Inco-ordination of the movement is also rare except after hemiplegia. Insular sclerosis of the brain and cord is attended by jerky inco-ordination, sometimes wild in its irregularity and in the extent of its range. It is important to know that a precisely similar form of inco-ordination is sometimes met with in cerebral tumor. I have seen it in cases of tubercle of the pons Varolii and crus cerebri, when the tumor has compressed the motor tract. There is usually hemiplegic weakness; and the movements of the leg, as well as those of the arm, may be irregular. Charcot believes that the mechanism of this inco-ordination in disseminated sclerosis is an unequal morbid resistance in the nerve-fibres passing through a sclerosed area, and a similar effect may, conceivably, result from a compression by a tumor.

LECTURE VI.

SYMPTOMS (*Continued*): DISTURBANCE OF SENSATION—AFFECTIONS OF SIGHT.

GENTLEMEN,—From the motor symptoms that occupied our attention in the last two lectures, we pass to the corresponding disturbances of sensation. The best instrument for testing sensibility is a simple one, a quill pen—the feather for touch, and the point for pain. We will consider first the loss of sensation that is analogous to the motor palsy, and often attends it. Hemiplegia may exist without any sensory loss, or with every degree of this loss. Complete hemianæsthesia may exist alone, or may be combined with every degree of motor palsy. It is rare, however, for the two to exist in high degree: usually a considerable degree of one is combined with a slight degree of the other. I am speaking now and always of organic disease, and have nothing to do, beyond a passing reference, with the mysterious hemianæsthesia that occurs in some cases of hysteria.

The one-sided loss of sensibility may involve all forms of sensibility—of touch, pain, temperature—or may involve one more than the others. In its complete form it extends up to the middle line, the absolute loss, however, usually stopping short of the middle line by a centimetre or so. It involves the mucous membranes as well as the skin. It always depends on interference with the sensory path, the course of which I described to you (p. 13); but if the lesion is in the pons, the parts supplied by the fifth nerve escape. The most frequent seat of disease causing hemianæsthesia is the posterior part of the internal capsule, between the posterior extremities of the optic thalamus and lenticular nucleus. Disease here, you will remember, may involve the special senses on the same side as common sensibility, the affection of sight being, however, hemiopia, due to arrested conduction from the half of each retina. Although the destination of the sensory path seems to be the cortex under the parietal bone, hemianæsthesia only results from cortical disease when this is very extensive, and then (as the case related on p. 14 shows) the special senses may be involved, and instead of hemiopia we may have loss of sight in the eye on the anæsthetic side, and perhaps hemiopia also in the eye of the same side, if the disease involves the occipital as well as the parietal lobe (see p. 20, note).

The slighter forms of defect may be characterized, not by any absolute loss, but by slight qualitative change. A patient, for instance, often avers that there is a difference throughout one side, that a touch or a prick does not feel the same as on the other side, although there is no place at which the slightest touch is unfelt.

The second form of loss of sensation often accompanies hemiplegia. The loss exists chiefly or only in the limbs, and is greatest in, often confined to, the extremities. Tactile sensibility is lost in greater degree than sensibility to pain, and there is often an inability to recognize the position of the hand or foot, even when the skin is perfectly sensitive. Thus, the patient's eyes being closed, the hand and fingers are moved about, and finally held in some posture which the patient is asked to describe, or to imitate with the other hand. If there is no tactile loss, the fingers should be firmly grasped so that the direction of pressure may not inform the patient of the posture. This should be repeated several times, since too much weight must not be laid upon a single error, especially if the patient is unintelligent. It is remarkable that in some cases in which the patient can feel the slightest touch on the hand he has not the least idea of the posture in which it has been placed. This loss occurs in disease of the motor cortex. It depends, according to Munk, on the loss of the structures that subserve conceptions of movement. We cannot yet say, however, that it does not result from disease elsewhere. Indeed, some of you may remember a case of hemiplegia that was in the hospital a few months ago, in which this loss was as distinct as it can ever be in an unintelligent patient, and the cortex was intact, the lesion being confined to the internal capsule and corpus striatum. Hence the diagnostic value of the symptom as evidence of cortical disease is probably not great.

It is a curious fact that lesions of the brain occurring in infancy or early childhood seem never to cause permanent loss of sensibility, although they must sometimes involve the sensory path or centres. This fact shows that, in the growing brain, compensation by other parts, perhaps by the other hemisphere, is possible in regard to this function in very complete degree.

The hemianæsthesia that occurs in hysteria may be misunderstood and ascribed to organic disease, but careful attention to the other symptoms in the case will generally prevent error. Remember that in hysteria one-sided loss of sensibility, although usually complete, is not always so; the special senses, and even the head, may escape. If it is combined with motor palsy, the loss of feeling may be much greater in the weakened limbs than elsewhere. The risk of error is greater in such cases than it is when the hemianæsthesia is complete and the special senses are affected—vision as "crossed amblyopia." Perfect as is the correspondence of this sensory loss to that which may result from organic disease of the cortex, the slight risk of error that there is may be avoided if you remember that the symptom can be produced by organic disease only when this is very extensive—so extensive that its nature could not admit of question. If you doubt whether there is organic disease, you may feel sure that there is not. In the only situation in which a small lesion can cause hemianæsthesia with affection of the special senses (the posterior part of the internal capsule), the affection of sight is always hemiopia.

Perhaps the greatest risk of ascribing hysterical loss of sensation to an organic cause is in the cases of infantile lesion of the brain that I have just mentioned. I told you, in the last lecture, how prone the female subjects of such disease are to develop hysteria. If you are not aware that such lesions do not permanently impair sensibility, you may easily ascribe hysterical hemianæsthesia in such a case to the organic disease. It is one of the most subtle of the many traps that hysteria delights to set for the unwary diagnostician. I had myself to pay for my experience. Some years ago a girl came to me with infantile hemiplegia of the ordinary type, with shortened limbs, and mobile spasm in the arm. She complained of no symptoms suggestive of hysteria. On testing sensibility I found that the whole of the paralyzed side was anæsthetic; the special senses were blunted on that side, vision was much impaired, and color-vision was lost. Here, I thought, are two most interesting things: first, there is a lasting loss of sensibility from a lesion in the brain in infancy; and secondly, there is crossed amblyopia from organic disease. I made most careful notes, and told her to come again in a week. She did so; and on the paralyzed side sensation was now perfect, and all the loss of feeling had gone over to the other side! Only a few months ago, in one of the medical papers, I saw an account of a similar case, which was published as hemianæsthesia of organic origin.

Symptoms due to irritation of the sensory tract or centres are not uncommon in central disease. Tingling, formication, and the varied sensations called "numbness" are frequently associated with hemiplegia, as prodromata and as accompaniments. They are common both with and without actual loss of feeling. When the lesion occupies the motor centres or path, these sensations, like the anæsthesia in the same cases, occupy chiefly the extremities. Such sensations, extending over the whole of one side, up to the middle line, attend disease in or near the sensory path, and may herald or accompany hemianæsthesia. The anæsthesia may be transient, and the sensations persistent, no doubt because the lesion is so placed near the sensory path or centres as to irritate but not to interrupt them. In the same class of cases there may be persistent pain in the limbs, usually dull and wearying. The patient whose fields of vision are represented in Fig. 8 suffered severe pains of this character for many years, the result of a lesion which caused transient hemiplegia and persistent hemiopia.

Paroxysmal sensations of tingling, "pins and needles," or pain, may result from such discharges as, in motor structures, cause convulsions, and the sensory discharge often accompanies the motor. I spoke of these sensations in the last lecture. It is also very important for you to know and remember that such one-sided sensations of tingling, etc., may occur as part of the strange phenomena of migraine. They are then more deliberate than those that herald a convulsive attack or constitute a minor seizure, lasting for half an hour or an hour. When on the right side, aphasia often attends them. Followed, as they are, by violent headache, they may cause much alarm, not only to the patient, but also to his medical adviser, if the latter is unaware of the occurrence of such sensory disturbance in this association. If the patient has not had such an attack before, he is certain to have suffered from paroxysmal headache, and probably from slighter sensory symptoms, especially in vision; from these you may feel sure of the nature of the attack.

We pass from the disturbance of common and tactile sensibility to the symptoms that depend upon disturbance of the nerves of special sense. These are cranial nerves, and it will be most convenient to go through the cranial nerves *seriatim*, considering the symptoms within the range of their functions in the anatomical order, which is nearly the order we adopted in the description of the anatomical relations of the nerves. Those relations you must bear in mind, if you would understand the important symptoms that are produced by disease of the nerves, or of the central tracts that continue their path to the cortex.

We commence, then, with the nerve of smell. Remember that the olfactory nerve subserves, not only the sensations that we recognize as odors, but also those that are termed flavors. We call the sensation an odor when its cause reaches the olfactory membrane by the anterior nares, a flavor when its cause enters the nose by the posterior nares. In the latter case the sensation is combined with a true gustatory sensation (of sour, sweet, bitter, etc.) from the tongue and fauces, and the two are so blended that we seem to taste flavors when we really only smell them. Hence those who have lost smell always say that they have also lost taste, although you will find, on trial, that the true gustatory sensations are unimpaired. In examining the sense of smell you must employ only odorous substances that are not pungent; *i.e.*, do not irritate the fifth nerve, or the patient may perceive that which he cannot smell, and the result is confusing. Loss of smell is an infrequent symptom of brain disease. Its most common cause is disease of the olfactory mucous membrane, polypus, chronic catarrh, and the like, the absence of which must always be ascertained before the symptom is allowed significance. Mechanical injury, by damaging the delicate filaments that come from the olfactory bulb, is another occasional cause. Diseases of the anterior fossa of the skull, tumor, aneurism, meningitis, sometimes cause it, and it may result from the pressure on the bulbs in extreme internal hydrocephalus. From central disease it is very rare, except in association with complete hemianæsthesia; it is then on the side opposite to the lesion. It probably may be produced also by disease in the temporo-sphenoidal lobe, near the inner part of the fissure of Sylvius, and will be then on the same side as the lesion. It occurs occasionally in degenerative diseases, as general paralysis of the insane, perhaps in consequence of an atrophy of the nerve analogous to that of the optic nerve. Subjective sensations of smell are almost confined to functional diseases (as the aura of epilepsy, etc.), but have been noted from tumor of the temporo-sphenoidal lobe.

Disturbed function of the optic nerve is a very common symptom of brain disease. In acute lesions it is generally due directly to the cerebral lesion; in chronic disease it is often the result of optic neuritis, and is thus a secondary effect of the brain-mischief, of significance chiefly as to the nature of the disease. In almost all cases, however, an ophthalmoscopic examination is necessary to ascertain whether the affection of sight is of intra-ocular origin, before its significance can be determined. There is only one form of impairment that can be referred with certainty to central disease, and that is hemiopia. Even in acute lesions of the brain, which do not, as a rule, entail optic neuritis, coincident retinal changes, such as albuminuric retinitis, hæmorrrhage, and embolism, sometimes cause impairment of vision, the origin of which can also only be ascertained by the ophthalmoscope. If optic neuritis is found, we are not justified in immediately ascribing the impairment of sight to its effect. If the visible neuritis is slight in degree it will not account for affection of sight. The latter may be due either to a more considerable retro-ocular inflammation of the nerve, or to the central disease, and between the two we can only decide by the symptoms.

I have already mentioned the chief points in the anatomical relations of the optic nerves that determine the form of loss of sight from disease in the several parts of the visual path. Loss of sight of one eye means an affection of its nerve between the eye and the chiasma, *i.e.*, in the orbit, at the optic foramen, or within the skull. Temporal hemiopia—loss of the outer half of each field of vision—means damage to the middle of the optic chiasma, affecting the fibres that cross from the nasal half of each retina, and leaving unaffected those at the sides of the chiasma that do not cross. Its most common cause is a tumor, distention of the third ventricle, and inflammation. Affection of only the non-decussating fibres, by damage to each side of the chiasma, causes nasal hemiopia, but is extremely rare. Disease anywhere in the optic path, from the chiasma to the occipital cortex, causes hemiopia, which may be complete or incomplete, but is always symmetrical, although not always minutely symmetrical. When incomplete, a part only of each half-field is blind. Amblyopia of one eye, with considerable peripheral restriction of the field of vision, occurs from extensive disease of the cortex of the opposite hemisphere, and perhaps from a small lesion in the angular gyrus. As I have told you (p. 21), there is usually loss of color-vision, and there is also slighter restriction of the fields (white and color) in the eye on the same side as the disease. When caused by acute non-progressive lesions, this loss usually soon lessens, perhaps by compensatory action of the centre in the other hemisphere, or of the undamaged part of the affected centre. Thus, affection of sight of one eye may be due to disease at either extremity of the visual arrangement—the optic nerve, or the highest cortical centre. The two are distinguished by the following indications: (1) the reaction of the pupil is lessened in disease of the nerve, and not in disease of the hemisphere; (2) the other eye suffers in a similar but slighter way in cortical disease; and (3) the associations of the two are wholly different, cortical disease being accompanied by hemianæsthesia and no affection of the cranial nerves; whereas, in disease of the optic nerve, hemianæsthesia is always absent, and the motor nerves to the eyeball are very often involved. Remember, however, that for one case of crossed amblyopia and hemianæsthesia from organic disease, you will see many that are of functional origin, and the evidence of an organic lesion must be very clear before you can ascribe the symptom to this cause.

A simple peripheral restriction of the field of vision occurs also in optic nerve atrophy, both in simple atrophy and in that which follows neuritis. In the latter the fields are often irregular; sometimes there are islets of loss ("scotomata," as they are termed) within the field, and such islets may vary for different colors. A central defect is a consequence of damage to the fibres in the axis of the nerve. It is common in tobacco-amblyopia, but is scarcely ever met with in brain disease.

In searching for these defects of sight you should first ascertain whether there is any considerable error in the refraction of the eye. A slight amblyopia, which you might otherwise ascribe to an affection of the optic nerve, may be due to this cause. It is beyond my present task to describe the methods by which the examination may be made with precision, but for the purpose in view a rough estimate may be formed in the course of the ophthalmoscopic examination. When you throw the light into the eye, as for the indirect examination, if you can see the retinal vessels distinctly, the refraction is abnormal: move your head to one side, and the vessels, thus seen, appear to move: if this movement is in the same direction as your head, the eye is hypermetropic; if in the opposite direction, the eye is myopic. You may further ascertain the degree of each defect in the direct ophthalmoscopic examination, by observing the strength of concave glass necessary for clear vision of the fundus if the eye is myopic, and the greatest strength of convex glass that does not blur the image if the eye is hypermetropic. Of course if your own eye is of abnormal refraction you must allow for this. In the further examination of vision each eye must be separately tested. Acuity of vision is ascertained by test-types, numbered according to the distance in feet (or metres in the case of Snellen's test-types) at which the type should be distinct. The result is expressed in the form of a fraction, of which the denominator is the number of the test-type, and the numerator the furthest distance at which the type is read by the patient. Thus, $\frac{1}{6}$ means that the type which should be read at a distance of six feet can only be read at one foot. Color-vision is examined by the method of comparison (with wools, etc.), for an account of which I must refer you to books on ophthalmology. It may also be tested by asking the patient to name colors—a method chiefly valuable when you wish to compare the color-vision of one eye with that of the other. By the method of comparison you escape the errors arising from the common inability to name colors with precision, and from the uncertainty of the natural appreciation of colors, and of the sense attached by the patient to the names he gives. This element, which we may term the personal color-equation, does not come in when we merely compare the sense-impressions of one eye with those of the other, and we may therefore, in this case, ask the patient to name the colors, and often learn much regarding his perception of color that can be ascertained in no other way.

The examination of the fields of vision is of especial importance in the diagnosis of brain disease. For an exact examination it is necessary to use a "perimeter," an instrument in which an arm, in the form of a quadrant of a circle (graduated in degrees), rotates on one extremity, and thus may be made to occupy successively the various radial arcs of an imaginary hemisphere, of which its pivot is the centre. The patient's eye being opposite and looking at the pivot (his other eye being closed), a small white or colored object is moved along the arm in each radial position, and the point at which the object ceases to be seen is the limit of the field in that direction. The results are marked on a chart, which consists of radial lines (corresponding to the positions of the arm), and concentric circles (corresponding to the divisions on the arm). A line joining the points in each radial line, at which the object ceases to be seen, indicates the peripheral limit of the field of vision. This, as already stated (see p. 17), is not a circle, being less extensive inwards than outwards, and above than below, on account of the projecting nose and eyebrows, and the influence of these on the functions of the corresponding parts of the retina. In Fig. 13, which represents an average normal field, the circles and radii are not represented beyond the limits of the field, the circles and radii may be as numerous as you like; in the figure only those at 30° degrees distance apart are represented. The size of the object may be a centimetre or half a centimetre square. To ascertain the color-fields, a colored object must be used, and the points noted at which the patient ceases to see the color, not the object. The latter can still be seen outside the region in which the color is recognizable, the fields for color being smaller than that for white, and their order, beginning with the smallest, is violet, green, red, yellow, and blue. An examination with the perimeter requires time, and need only be employed when you have reason to suspect that there is a limitation of the field. You may ascertain whether there is any considerable defect by a much rougher method of examination. Let the patient stand opposite to you, and about two feet away. Make him place his hand over the eye you do not wish to examine, and keep the other fixed on your eye that is opposite his, your other eye being closed. Next place your hand, midway between yourself and him, in various places towards the limit of your own field of vision, and ask him if he sees it; move your fingers, if you like,

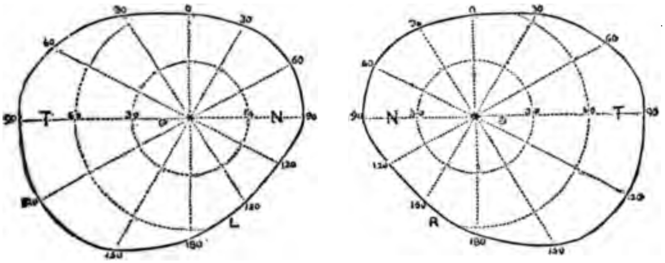

FIG. 13.—DIAGRAMS OF THE NORMAL FIELDS.

The asterisk is at the fixing point; the small circle indicates the blind spot (optic nerve entrance); R, right; L, left; T, temporal or outer side; N, nasal or inner side. The radii and concentric circles are each 30° apart.

and ask him if he sees them move. Still better, place a small piece of white paper on the end of a dark-colored stick (a penholder answers well) and hold the paper in various positions all around the outer part of the field, or nearer the centre if you suspect any central loss of vision, holding it so that the light falls well upon it, and the surface, not the edge, is towards the patient. In this method the patient uses your own eye as a fixing point, and you are able to see at once if he looks away from the fixing point and at the object, as he is almost sure to do at first. You check his field of vision by your own, and do not move the object into positions in which it is beyond the natural range of sight.[1] A still rougher method of ascertaining that there is no symmetrical hemiopia is the following:—Let the patient stand opposite to you with both eyes open, looking at you. Hold up both your hands, one on each side, as far apart as you can well see them. Then put them down and ask him if you held up one hand or two. Do not ask him this while the hands are up, or he is almost sure to look first at one and then at the other, and say "two." If both hands were seen there is no defect in the outer half of either field, and therefore no ordinary hemiopia, since, in this, one outer half must be defective.

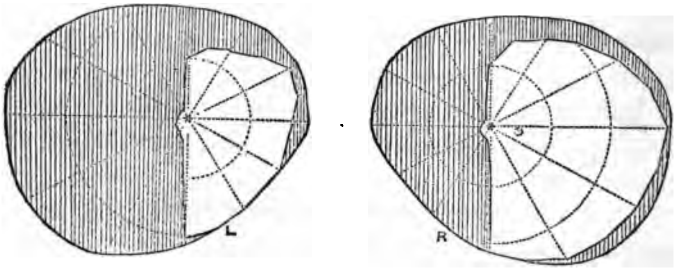

FIG. 14.—FIELD OF VISION IN A CASE OF LEFT-SIDED HEMIOPIA.

The shading represents the blind part; the oval outline of each figure is the average normal field. The asterisk is the fixing point.

Remember that the subjects of hemiopia are not always conscious of it; especially when the line of blindness does not go very near the fixing point, the loss is often unnoticed. One woman with such hemiopia was indignant at the suggestion that she had any defect of sight, declaring that she "could see her husband coming across the common before any one else could." In another patient the defect was not discovered until the nurse noticed that the man never ate his potatoes at dinner. They were always put on the same side of his plate, and he never saw them. The same patient once demonstrated his hemiopia to a gentleman in the street, who was walking with a lady. In the patient's effort to avoid the lady he walked right on to the gentleman. The subjects of this defect often hold the head inclined towards the blind side, so as to bring objects further into the remaining part of the field, and thus see the relations of objects better. A hemiopic cabman was thus able to drive his hansom about London quite well. But if he wanted a fare he had to go on a rank, because if he looked about him, he was sure to run into something.

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[1] For testing the color-fields in this manner, Messrs. Pickard & Curry make small colored metal discs, that can be fixed on a pen, and carried in an ophthalmoscope case.

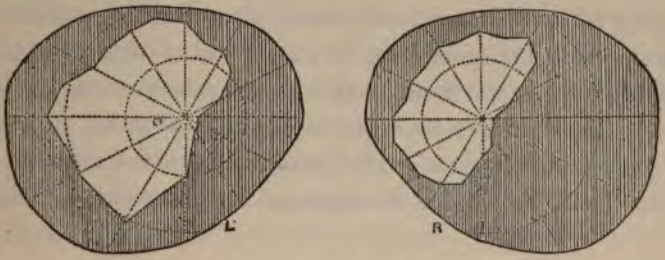

FIG. 15—RIGHT HEMIOPIA FROM CEREBRAL DISEASE, SHOWING AN IRREGULAR OBLIQUE LINE OF DIVISION, PROBABLY DUE TO AN UNUSUAL FORM OF DECUSSATION.

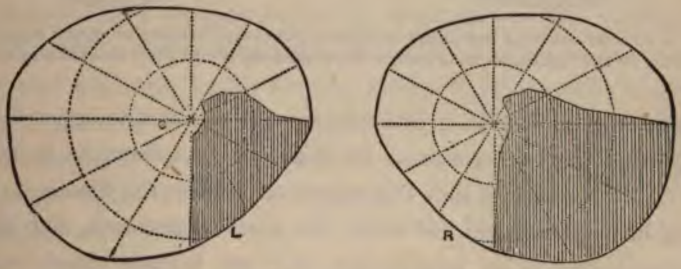

FIG. 16.—PARTIAL RIGHT HEMIOPIA FROM DISEASE OF THE LEFT CEREBRAL HEMISPHERE, THE LOSS BEING NEARLY QUADRANTIC. (Note the close correspondence of the shape of the blind area in each field.)

The chief facts regarding the form of hemiopia are five, and I have already alluded to some of them in speaking of the indication they afford of the relation of the optic path. The first is that the division may pass through the fixing point, or may diverge so as to leave it within the region of vision (Fig. 13). Secondly, the line of division is often slightly irregular: the divergence that leaves the fixing point in the region of vision may commence just above, and end just below the fixing point; or may begin some distance above and extend below this. Thirdly, the line of division often inclines to one side or the other, above and below (as in Figs. 14 and 17), and this inclination may be in opposite directions above and below, so as to give rise to an oblique hemiopia (Fig. 15). All these variations are probably due to individual variations in the decussation of the optic nerves, and not to the seat of disease. The next fact is that the hemiopia may be incomplete, only a portion of each half being lost. It is usually a segmental defect; often an irregular quadrant, as is shown in Fig. 16. This partial loss is due to partial destruction of the visual path or centre, and depends on the limited extent of the lesion (see p. 19). Lastly, the hemiopia may be accompanied by concentric restriction of the remaining half-fields, greatest in the eye on the side opposite to the lesion, and sometimes confined to this eye. An instance is shown in Fig. 17. It is probable that this occurs only when the disease is in the cerebral hemisphere, and not when the tract is diseased, and that the restriction is due to an interference with the highest visual centre, or with the fibres passing to it. The restriction is not always present even when the disease is in the hemisphere. You must remember, however, that concentric restriction of the fields may result from optic neuritis, and it may then be even greater than that which results from the cerebral lesion. The only difference between the characters of the two is that the restriction from neuritis is more nearly equal in the two eyes than is that from central disease.

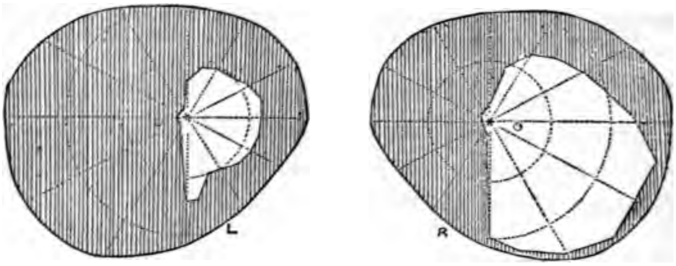

FIG. 17.—LEFT HEMIOPIA FROM DISEASE OF THE RIGHT CEREBRAL HEMISPHERE, WITH CONCENTRIC RESTRICTION OF THE REMAINING HALF-FIELDS, MUCH GREATER IN THE LEFT EYE THAN IN THE RIGHT.

Transient loss of sight occurs in some functional diseases of the brain, but its paroxysmal character precludes almost all danger of mistake.

In epilepsy the loss is complete, but is usually followed instantly by loss of consciousness. In migraine, hemiopia is common, and total loss is rare, but the symptom lasts, as a rule, only a quarter or half an hour. The affection of sight that occurs in hysteria I have already mentioned; there is usually considerable amblyopia of one eye, with slighter amblyopia of the other, and extensive impairment of the other special senses and cutaneous sensibility on the side of greatest visual loss. The distinction I have just described to you. In hysteria there may be also complete loss of sight of one eye, the other being unaffected; but this is rare. The perfect action of the pupil sufficiently distinguishes it. It is doubtful whether hemiopia is ever of hysterical origin; and this is strange, because, in migraine, hemiopia is common. Apparently the two affections are on different functional levels, and keep to them. Persistent hemiopia is almost always due to organic disease, and its significance is therefore decided. Of a large number of cases of hemiopia of which I have notes, there is only one which I think may have been hysterical.

Irritation-symptoms in the functions of the optic nerves are rarely due to organic disease, while they are common in functional disturbance. Intolerance of light occurs in meningitis, but is rather a cerebral than an ocular symptom—light seems to distress the head rather than the eye. True photophobia is scarcely known in brain disease, or in diseases of the optic nerve. Visual discharges, subjective lights or colors, occur in epilepsy and migraine; in the former, flashes or points of light are common; in the latter, the zigzag "fortification" appearance is characteristic. Disease near the visual centre in the cortex may cause subjective sensations of light, but only as symptoms of a discharge that quickly bursts into a convulsion.

LECTURE VII.

SYMPTOMS (*Continued*): OCULAR NERVES—FIFTH NERVE—FACIAL NERVE.

GENTLEMEN,—We may conveniently consider together the symptoms that depend on the three cranial nerves that supply the eyeball muscles—the third, fourth, and sixth. All supply external muscles; the third nerve also some internal muscles. Paralysis of the external muscles is indicated by symptoms of five kinds: (1) limitation of movement; (2) non-correspondence of the direction of the two eyes, *i.e.*, strabismus; (3) double vision; (4) erroneous projection of the field of vision of the paralyzed eye; (4) secondary deviation of the unaffected eye. The defect of movement is always in the direction of action of the paralyzed muscle, and is termed the "primary deviation." It is most conspicuous when the unaffected eye "fixes" the object, and the affected eye is prevented seeing it by the interposition of the hand, or a piece of paper, in such a manner that the eye can still be observed. The "secondary deviation" is an excessive movement of the sound eye, when *this* is prevented seeing the object at which the affected eye looks. If then the interposed hand be moved so as to cover the affected eye, the sound eye, to "fix" the object, has to move back again, and the degree of backward movement indicates the degree of previous excess, that is, of secondary deviation. Both primary and secondary deviation depend on the circumstance that the muscles of the two eyes reserve an equal amount of innervation from the centre. That which suffices to move the sound eye into a given position is insufficient to move the affected eye into the corresponding position, and the innervation necessary for *this* causes an excessive movement of the sound eye. Thus, the primary deviation being a defect, the secondary deviation is an excess; they are in opposite directions, but both are in the line of action of the affected muscle. In slight palsy the secondary deviation is often more readily observed than the primary deviation. The patient habitually fixes with the unaffected eye, and hence the primary deviation, the paralytic defect of movement, occurs whenever the eyes are so moved as to bring the affected muscle into action. The difference in the direction of the two eyes constitutes strabismus, and from it there results double vision. The paralytic strabismus may be convergent or divergent, or there may be a difference in the height of the two eyes, according to the muscle affected. The strabismus is distinguished from that due to muscular spasm by the fact that the latter is the same in all positions, whereas the former only occurs in the positions necessitating the action of the affected muscle; in spasmodic strabismus no secondary deviation can be obtained. Double vision is commonly present in paralysis, at least when this is recent, and absent in the common spasmodic strabismus. This difference does not depend on the nature of the two affections, but on their duration. In chronic deviation of the eyes, from whatever cause, the patient learns to neglect the image seen with the affected eye (termed the "false image"), and to attend only to that seen with the sound eye ("true image"), and may be quite unaware of a diplopia which is at once conspicuous to him if a colored glass is placed before one eye so as to tint one of the images. It is best to place the colored glass before the sound eye. When the images are so near together that they overlap, the image appears to be blurred rather than double, but the colored glass distinguishes the two. If a colored glass fails at first to reveal the double image, the observer's hand may be placed before first one and then the other eye, and the object will appear to change its position as well as its color with the change in the eye obscured. Double vision may be "crossed" or "simple." In crossed diplopia the image in the left eye is seen to the right of that in the right eye. In simple diplopia (usually called "homonymous" or "same-named") the image in the left eye is seen to the left of the other. When the strabismus is divergent, the diplopia is crossed; when convergent, the diplopia is simple. If you will make a diagram for yourselves of the eyes in the two positions, and of the rays of light from an object, you will understand this without difficulty. Should you find it hard to keep in mind the character of the diplopia in the two cases—and these simple alternatives often give much trouble to the student—remember that when the prolonged axes of the eyes would cross, the double vision is *not* crossed.

The recent paralytic diplopia exists only in positions of the eyes needing the contraction of the weak muscle, and the farther the object is moved in the direction of action of the muscle, the farther apart are the two images. In old-standing cases some secondary contracture of the antagonists may give rise to diplopia in other positions, and even at rest. In many positions of the eyes the two images are not parallel. This depends on the complex associated action of oblique and straight muscles, with which you are doubtless already familiar. If a paralyzed straight muscle, in a certain position, has a tendency to rotate the globe, which tendency is normally counteracted by one of the oblique muscles, the latter producing its effect alone, causes an abnormal rotation of the eye.

The "erroneous projection of the visual field" is not quite so abstruse a subject as some old associations with the word "projection" may lead you to fear; and it is a question of much interest. We judge of the relation of seen objects *to each other* by the relative position of their images on the retina, *i.e.*, by their relative position in the visual field; but we judge of their relation to our own body by the relation to it of the whole visual field. This depends on the position of the head and of the eyes, and we know *this* by the degree of innervation of the muscles that move the head, and especially of those that move the eyes. We are only now concerned with the latter. When the innervation of the ocular muscles is at a minimum, the eyes are in mid-position, and we know, to use popular language, that an object at which we are looking is opposite our face; that is, that a line from the centre (fixing point) of the field of vision to the macula lutea would stand at right angles to the plane of the face. If we turn the eyes to one side, we know that an object *now* in the centre of the field is to the side of the position of the former one; how far to the side, we judge by the degree of movement of the eyes, estimated by the amount of innervation that we have given the muscles. I say "we judge," but there is no conscious judgment; the estimation is by nerve-processes of which we are unaware. If we want to touch this second object, we adjust, also unconsciously, the degree of contraction of the muscles of the arm, so as to enable us to effect the required movement with instant precision. But if the eye muscle concerned is weak, the increased innervation necessary for the movement gives an impression of a greater movement than has really taken place, and therefore an impression that the object looked at is farther on that side than it really is, and if the patient attempts to touch the object, his hand goes beyond it. His arm moves, in accordance with the innervation of the weak muscle, too far in the direction of the action of this muscle, just as the other eye moved too far in the secondary direction. Indeed, the erroneous direction of the arm corresponds exactly with the direction of the secondarily-deviating eye. But the knowledge of the relation of external objects to the body, gained from the innervation of the eye muscles when we look at the objects, is one of the most important guides to the centres that regulate the maintenance of equilibrium. The erroneous projection of the field of vision causes a discord between this and other guiding sensations, and one effect of this discord is the sensation that we call vertigo or giddiness. This giddiness, depending on paralysis of an ocular muscle, is termed "ocular vertigo," and, like other forms of vertigo, is apt to cause mistakes in diagnosis. The nerve-centres, after a time, learn to accommodate their action to the altered circumstances, and both the erroneous projection and the consequent vertigo cease to trouble the patient. When it exists, the patient may try to avoid it by holding his head in such a position as not to call the affected muscle into action, and this will often put you at once upon the track of the affection. Or he may keep the affected eye closed, by contracting the orbicularis. He always closes the affected eye, because, although closing either eye would remove the diplopia, only closure of the affected eye removes the vertigo or uncertainty. He may thus show you which eye is at fault, when you might find it difficult to ascertain. If you cultivate the habit of observing every point, however trifling it may seem, you may often learn much of the nature of a patient's ailment before he has got half-way across your room.

I may briefly summarize for you the chief symptoms of the paralysis of each muscle. I will not ask you to try to remember them now, but it may be convenient to you to have the summary for future reference. We will take, as examples, the muscles of the right side.

Sixth Nerve.—External Rectus.—Defect of outward movement, convergent strabismus on looking to the right, with simple diplopia, the two images parallel on the same level in the horizontal outward movement, but the false image often on a different level, and oblique, when the eyes are directed above or below the horizontal level. Secondary deviation of sound eye inwards. The head is inclined to the right.

Third Nerve.—Internal Rectus.—Defect of movement inwards, *i. e.*, on looking to the left, with divergent strabismus, and crossed diplopia, the false image oblique above and below the horizontal plane. The head is inclined to the left.

Superior Rectus.—Defect of movement upwards, and especially upwards and outwards; the inferior oblique, which habitually acts with the superior rectus, being capable of aiding the upward and inward movement, it may visibly rotate the globe in so doing. Secondary deviation is by the opposite superior rectus moving the eyeball too much upwards. Diplopia occurs on looking upwards; the false image is above the other, and the difference in level is greatest in trying to look upwards and to the right.

Inferior Rectus.—Defect of movement downwards, and especially downwards and outwards (because the downward and inward movement is aided by the superior oblique). There is corresponding double vision, which is crossed. The false image is below the others, and the lower the greater the attempted movement; it is also oblique, especially on the inward movement, from the rotation effected by the superior oblique. Secondary deviation is by the opposite inferior rectus.

Inferior Oblique.—Defect of movement upwards, in which position the muscle is an elevator (just as the superior oblique, in the inward and downward movement, is a depressor). The secondary deviation is also upwards and inwards, by the inferior oblique and the internal rectus of the sound eye. Double vision exists in the same movement; the false image is above the other, and oblique, the obliquity being greatest in looking outwards, the difference in height greater in looking inwards.

Fourth Nerve.—Superior Oblique.—Defect of movement, chiefly downwards and inwards, since in this position the muscle is a depressor. Convergent strabismus exists in looking down, and the corresponding diplopia is simple, the false image being the lower, especially in an inward movement, and oblique, especially in the outward movement; the lateral distance between the two is greatest in the middle line, and lessens when the object is moved inwards or outwards. The diplopia, occurring on looking downwards, often gives the patient much trouble in going down a flight of stairs, which seems to be double, and he does not know which flight to choose.

You must not imagine, however, that it is always possible to determine with exactness the muscles that are affected in the derangement that results from brain disease. It is generally possible when the nerves themselves are diseased, but in central affections there is often a complex association of palsy and spasm that may baffle all attempts to unravel it. I remember, some years ago, one of the most distinguished ophthalmic surgeons in Europe, who knew, perhaps, more of ocular palsies than any one at that time living, visited the Queen Square Hospital and examined some patients for us. After spending some time in the investigation of the defect of movement of the eyes in one case, he said: "I can tell you many things that it is not, but I cannot tell you what it is."

The special symptoms of paralysis of each ocular nerve need not detain us long, since those of the sixth and fourth nerves are the palsies of the muscles they supply, the external rectus and superior oblique. When the whole third nerve is paralyzed all the muscles are affected except the two just named; the eyeball cannot be moved upwards or inwards, and only slightly downwards, while the unopposed action of the external rectus usually causes the eye to deviate outwards. There are, however, three other symptoms: the upper lid droops from the paralysis of the levator (ptosis); from the paralysis of the sphincter of the iris, the pupil is midway between contraction and dilatation, and does not contract to light; while the paralysis of the ciliary muscle abolishes the power of accommodation.

Isolated paralysis of these muscles is usually due to disease of the nerves in the pons, in the crus, at the base of the brain, in the orbital fissure, or in the orbit; but curious associated palsies are sometimes met with in central disease. Loss of the upward or downward movements and of convergence (without loss of other action of the internal recti) has been observed, and is probably due to a lesion in or near the third nerve-nuclei or in the corpora quadrigemina, usually degenerative in character. Loss of the movement of both eyes to one side occurs in focal lesions of the brain, and has been already mentioned (p. 46). I will only now tell you further that this "conjugate deviation," as it is termed, may be the result of either paralysis or spasm. In disease of the hemisphere the eyes deviate in paralysis towards, and in spasm from, the side of the brain affected. But in disease of one side of the pons they may deviate in palsy from the side of the lesion. Sometimes first one and then another ocular muscle becomes paralyzed until all have lost power, and the eyes are motionless, with a peculiar unchanging stare. There may also be ptosis. This affection—the progressive ophthalmoplegia of v. Graefe, the ophthalmoplegia externa of Hutchinson—depends on a degeneration of the nuclei of the muscles, analogous to that which, in the cord, causes progressive muscular atrophy in the limbs, and in the medulla oblongata gives rise to what is termed "progressive labio-glossal paralysis."

The muscular mechanisms within the eye are susceptible of four forms of paralysis. First, the ciliary muscle may be paralyzed, causing loss of accommodation, so that objects cannot be seen well, except at a distance, and small print, that could only be read near, cannot be read at all. The contraction of the iris associated with accommodation may be lost, either alone or with accommodation. The reflex contraction of the iris, on exposure to light, may be lost alone, and so also may the reflex dilatation on stimulation of the skin. Combinations of these palsies often occur, especially of the associated action of the pupil and of the ciliary muscle, and of the light and skin reflexes. Loss of accommodation may also be combined with loss of convergence. Sometimes all the internal muscles are paralyzed—the ophthalmoplegia interna of Hutchinson. These internal palsies are rarely the result of focal lesions. They result from influences acting on the nerve-elements according to their function. Thus, in diphtheritic paralysis the ciliary muscle especially suffers. Apart from diphtheria the chief cause of these affections is degeneration, usually associated with signs of degeneration elsewhere. This is their chief diagnostic indication, and a very important one it is.

Paralysis of the fifth nerve may involve the sensory or the motor parts, or both. The symptom of the former is anæsthesia of the skin from the vertex to the lower jaw, and of the mucous membrane of the nose, tongue, and mouth on that side. Ammonia and snuff no longer irritate the nasal membrane, but odors can be perceived, although, after a time, the sense of smell is blunted from defective secretion. When the patient drinks, the cup, felt only on one side, seems broken. Food is not chewed on the affected side because it cannot be felt, and often because the muscles of mastication are also paralyzed: hence fur accumulates on that half of the tongue, as it does whenever food cannot be chewed on one side. There is much discrepancy of evidence as to the influence on sensation at the back of the tongue and palate. In some cases of disease limited to the root of the fifth nerve there is anæsthesia of these parts, including the soft palate and anterior palatine arch. In other cases these parts have been sensitive. We do not know whether the difference depends on personal variations or on the position of the disease: probably on the latter. Similar discrepancies exist with regard to the affection of taste, as I have already told you (see p. 22). Taste is usually lost on the front of the tongue, and is certainly sometimes lost everywhere, even on the palate and its anterior arch. The probable explanation of this I have already given you. Trophic changes may result from paralysis of the sensory part of the fifth. Of these the most important is that in the eye. The cornea becomes cloudy, then opaque; ulcers form upon it, and may perforate, and lead to a destructive inflammation of the globe. This "neuro-paralytic ophthalmia," as it has been termed, probably depends on the irritation of the nerve by the lesion rather than on the anæsthesia or on the mere loss of nerve-influence, and the irritation seems to be most effective when it involves the Gasserian ganglion or the fibres from the ganglion in the nerve in front of it. The symptom is rare when the disease is situated between the ganglion and the pons, and the lesion there has to be more irritative in character to produce the effect.

Paralysis of the motor part of the nerve causes weakness of the masseter and temporal muscles, easily recognized if you make the patient "bite hard" while your fingers are on the muscles on each side. If the loss of power is slight, the weaker muscle seems to contract a little after the other. The paralysis of the external pterygoid causes a defect in the lateral movement of the jaw; this can be moved towards the paralyzed side, but not from it. When the lower jaw is depressed, it deviates towards the paralyzed side, because, in depression, the external pterygoids draw the condyle of the jaw forwards; and this movement, occurring only on the unaffected side, causes a deviation. The movement of the hyoid bone is not perceptibly impaired; and although the tensor palati and tensor tympani are said to be supplied by the fifth, they do not seem to suffer when the root is diseased, and hence, perhaps, their fibres come ultimately from some other source. After a time the paralyzed muscles waste, the temporal and zygomatic fossæ become flattened, and ultimately a little secondary shortening of the muscles may limit, in slight degree, the downward movement of the jaw.

Paralysis of the face is the result of disease of the fibres or nucleus of the facial nerve, or of the motor path between the facial nucleus and the cortex. This, as we have seen, lies to the inner side of the limb-path in the crus, and in front of it in the internal capsule, occupying the angle at the junction of the anterior and posterior parts of the capsule. The loss of power is on the same side as a lesion of the nerve-fibres, or of the nucleus in the pons; but, since the upward path decussates just above the nucleus, a lesion of the upper part of the pons, of the crus, or of the hemisphere, causes paralysis of the face on the side opposite to the lesion. There is another difference between the effect of disease above the nucleus and that of or below the nucleus. In the latter case all parts of the face are affected; the eye cannot be closed, the forehead cannot be wrinkled, and the mouth can neither be widened nor firmly closed, the one half of the orbicularis being paralyzed. Hence the patient cannot whistle, and he cannot "puff" out a candle, for which complete closure and sudden opening of the lips are requisite. But in the case of a supra-nuclear lesion the upper part of the face is little affected, and the orbicularis is scarcely weakened, the chief palsy being that of the zygomatici and elevators of the upper lip. The explanation of this has been already given in the account of hemiplegia, of which such palsy usually forms part. Another important difference is presented by the electrical reaction. In nuclear, and infra-nuclear disease, the nerve-fibres degenerate, and the nerve-trunk, in severe cases, can no longer be stimulated by any form of electricity. The muscles no longer respond to faradaism, since the intra-muscular nerve-fibres, on which only faradaism acts, are degenerated; but the muscular fibres still contract to the more deliberate stimulus of a slowly interrupted voltaic current, and even act to this with greater readiness than in health. This is termed the reaction of degeneration. I have explained its characters more fully in speaking of the diagnosis of diseases of the spinal cord. In the complete form of facial paralysis, the loss of muscular tone and action produces a strange effect on the aspect of the patient. In the young, in whom the elasticity of the skin largely moulds the features, the paralysis is little noticeable at rest, but is grotesquely obtrusive on any movement of the face, one side of which may be stern and unbending, while the other is convulsed with laughter. In the old, however, the skin is inelastic, and is thrown into wrinkles by the contracture in which the muscles stereotype the emotions they have habitually expressed. When the muscles become toneless, the wrinkles disappear, and the palsy reproduces the unseared features of an earlier age. The contrast throws into such relief the effect of age, that patients often refuse to believe that the smoother half can be unnatural, and maintain that the wrinkles are the morbid appearance. Indeed you can never place reliance on the statements of patients or their friends as to the side of the face that has been affected. The paralysis of the zygomatici on one side permits their fellows on the other to draw the mouth over, and far over on emotional expression. This renders the error an easy one. I have often heard a patient say that the unaffected half of the face "must have been the side that was wrong, because the mouth was drawn right up to the ear." Physicians in their turn are liable to be misled in another way. In cases that do not recover perfectly a curious contracture occurs, and voluntary movements, while less in degree, spread too widely over the face, so that the eye closes unduly in smiling, and the corner of the mouth is drawn up when the eye is closed. This contracture deepens the naso-labial furrow; the normal side of the face may be the smoother of the two, and may be thought to be the paralyzed side, until the degree of movement is observed. This contracture, in the young, causes a deformity almost as serious as the earlier palsy, but in the old it has a cosmetic influence, bringing back the natural furrows, and rendering the face symmetrical. The common cause of complete palsy is neuritis of the nerve just within the Fallopian canal, or ear disease, damaging it in its course through the temporal bone, and you would not think of an intra-cranial cause (within the pons or at the base of the brain) unless there were other symptoms of such disease. When the nerve is affected at the lower part of the canal, the chorda tympani often suffers, and taste is lost in the front of the tongue. It is often said that in disease of the nerve higher up, the palate is paralyzed, but I doubt whether this is true. For fifteen years I have been looking out for paralysis of the palate in disease of the facial nerve, and I have never seen it. I am inclined to think that the opinion is due to a misinterpretation of the very common congenital obliquity of the uvula, and inequality of the palate.

LECTURE VIII.

SYMPTOMS IN THE REGION OF THE CRANIAL NERVES (*Continued*): AUDITORY; GLOSSO-PHARYNGEAL; PNEUMOGASTRIC; SPINAL ACCESSORY—PARALYSIS OF THE LARYNX AND OF THE PALATE —HYPOGLOSSAL NERVE—COMBINED PALSY OF THE BULBAR NERVES.

GENTLEMEN,—We will to-day continue and complete our study of the symptoms that are due to disturbance of the function of the cranial nerves. The auditory and bulbar nerves remain for consideration. The term "bulbar" is a convenient designation for those nerves that arise from the medulla oblongata. You will remember that the auditory, facial and sixth arise at the level of the junction of the pons and medulla, but the facial and sixth ascend to their nuclei. That of the sixth is altogether, and that of the facial in great part, above its surface attachment, while the auditory nuclei are at the level of origin of the nerve. The nerves below the auditory, the glosso-pharyngeal, pneumogastric, accessory part of the spinal accessory, and hypoglossal, arise from, and have their nuclei within, the medulla oblongata, and are therefore those included in the term "bulbar nerves."

Disturbances of function of the auditory nerve are occasionally important symptoms of brain disease, but they are so far more frequently due to derangement of the delicate and complex organ of hearing that the presumption is always in favor of an aural rather than of a cerebral cause. The symptoms of disturbance are three—deafness, tinnitus, vertigo. Deafness may be the result of impaired conduction in the nerve, or of disease of its centre. Tinnitus aurium, or "noises in the ears," is due to irritation of the nerve or its central or peripheral terminations. Vertigo results from interference with the fibres that are distributed to the semicircular canals, and subserve, not hearing, but perception of the position and movement of the head—the fibres that Cyon calls the "space nerve" (an inapt designation, although the only one that has been given to this important part of the auditory nerve).

The deafness that is due to disease of the auditory nerve is the same in character as that which is due to disease of the labyrinth in which the nerve-fibres end, but it is sharply distinguished from that due to impaired conduction through the tympanic cavity (middle ear) or external meatus. The distinction is that the latter impairs hearing through the air, but leaves unaffected the perception of sounds conducted through the bones of the skull. The former alters both alike. One mode of ascertaining this is with a tuning-fork, and the test depends on the fact that the hearing through the air is normally rather more acute than that through the bone. A vibrating tuning-fork is held in contact with some part of the skull, say the parietal eminence. As soon as it ceases to be audible it is removed from the skull, and held opposite the auditory meatus. It should be still distinctly heard. If it is not, there is impaired conduction through the meatus or middle ear, and the former can readily be excluded by the speculum. If the tuning-fork is still heard, any deafness must be of nerve origin, *i.e.*, due to disease of the auditory nerve or nerve-endings in the labyrinth. The watch affords another test that is of great practical importance, not merely on account of its convenience, but because it is even more delicate than the tuning-fork, since perception, through the bone, of the short high-pitched sound of a watch is, for some reason, often impaired out of all proportion to the loss to the tuning-fork. The meatus should be closed by pressing the antitragus over it, the watch held close to, but not touching, the zygoma, and the patient asked if he can hear it. It should then be pressed firmly on the zygoma. In health it can scarcely be heard in the former position, but the sound becomes loud as soon as it is pressed against the bone. This is the case also in disease of the middle and external ear. In disease of the labyrinth and nerve, it is common for the sound to be quite inaudible when the watch is in contact. When this is the case we can be quite sure that there is nerve-impairment. Indeed, a caution is necessary on account of the delicacy of the test. Many persons, especially after middle life, have lost the power of hearing a watch through the bone, when they are conscious of no impairment of ordinary hearing. This is no doubt due to pathological changes in the labyrinth, for the change is often one-sided, and many of these persons suffer from tinnitus or vertigo. If the sound is still heard through the bone, although not so loudly as in health, we cannot infer disease of the labyrinth or nerve, because simple anchylosis of the stapes will lessen perosseal hearing (since some vibrations pass through the chain of bones), but no disease of the middle ear will extinguish all hearing through the bone. Galton's whistle, by which a very high-pitched note is produced, may also be used to test the hearing of such continuous sounds, but the pathological significance of their loss has yet to be defined. Very rarely a limitation of hearing exists, analogous to the limitation of the field of vision. It probably occurs chiefly in degeneration of the optic nerve. Thus, a patient with locomotor ataxy and optic nerve atrophy, who doubtless has also atrophy of the auditory nerve, has gradually become considerably deaf to all sounds, and is absolutely deaf to the loudest musical notes above E of the treble clef, and below the lower G of the bass.[1]

Deafness from intracranial disease is usually due to disease of the nerve at the base of the brain, less frequently of its nucleus in the medulla. It very rarely results from disease of the auditory centre in the first temporo-sphenoidal convolution, and is then on the side opposite to the disease. Bilateral deafness may be due to damage to both auditory nerves, and I have also known it to be produced by a tumor of the corpora quadrigemina damaging the upper layer of the tegmentum of each crus cerebri, in which the auditory path lies. From symmetrical disease of the cortical centres it is extremely rare. But deafness on both sides is frequently due to symmetrical labyrinthine disease.

Subjective noise, tinnitus aurium, is, like deafness, usually aural in origin. It may result from almost any disease in any part of the ear. It may be due to an abnormal sensitiveness to the movements that are normal in the ear—of the blood in the vessels (especially in the internal carotid), or of the muscles within the tympanic cavity,—or it may be due to an abnormal increase of these movements, *e.g.*, to an increased vibration of the moving blood in anæmia, or in carotid aneurism. It may be due to pressure on the auditory nerve, or to irritation, functional or organic, of the auditory centres. Its characters are extremely varied. The first point to be ascertained is whether the sound is pulsatory or continuous. In the former case the pulsation will be found to be synchronous with the heart's action, and the sound probably has its origin in the ear. Such a sound has been known to be due to an aneurism, and to be audible on auscultation of the skull. This is the only case in which a murmur is so audible, and so the diagnostic significance of the phenomenon is great, although it is extremely rare. Sounds that are due to irritation of the labyrinth or nerve, or are of central origin, are usually (though not always) continuous, and not pulsating. The next important fact is the elaboration of the sound. Sounds that are of simple character, "rumbling," "buzzing," "hissing," "whistling," may be of either local or central origin, but those that are of high elaboration, such as bells or music, are generally, the latter always, of central origin. I have known the sound of bells ringing to be the aura of epileptiform convulsions due to a tumor beneath the first temporal convolution. We have little definite knowledge of the relation of tinnitus to disease of the auditory nuclei of the medulla, but the sound of a loud crash has attended the onset of acute lesions in the vicinity of the nuclei. Whether the sound is referred to the ears, or vaguely to the head, is of little diagnostic importance. Intense bilateral sounds of subjective origin are usually referred to the head, and not to the ear. Tinnitus and deafness are often associated, and must then be ascribed to the same cause. The vertigo that results from affection of the auditory nerve will be considered in connection with other forms of vertigo.

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[1] Since the above was written, the range of hearing has gradually become further restricted, until only the notes between the two E's of the treble clef can be perceived. Even the lower E flat is inaudible.

The glosso-pharyngeal nerve and the pneumogastric need not detain us long, since their isolated affection forms a less frequent factor in the diagnosis of brain disease than does a general derangement of their function. This may occur from a severe lesion in any part of the brain, but is most conspicuous when the disease is in the medulla oblongata. We will consider subsequently the chief symptoms that are due to derangement of the visceral functions of the pneumogastric, and deal now only with two effects of the impairment of these nerves—paralysis of the pharynx, and paralysis of the larynx. With these, however, we may, for reasons that will appear as we proceed, most fitly consider the paralysis of the palate.

The motor nerve-supply to the pharynx is derived from the pharyngeal plexus, into which both the glosso-pharyngeal and pneumogastric enter largely, and we do not know from which of these nerves the motor fibres come. In paralysis of the pharynx, swallowing is difficult; food is apt to lodge, or get into the larynx, and particles may even find their way into the lower air-passages, and, after a time, set up chronic disease in the lung. Paralysis of the pharynx is rarely an isolated symptom of brain disease, and its diagnosis is not difficult. I have known it to be mistaken for cancer, but an examination should prevent the error. Palsy is painless, while cancer rarely is, and the former is usually accompanied by loss of power in adjacent parts.

You will remember that the larynx receives only sensory fibres from the pneumogastric proper, its motor fibres coming entirely from that part of the spinal accessory that joins the vagus. You will remember also that the superior laryngeal nerve contains the sensory fibres for the vocal cords, and for the larynx above them, and motor fibres for the crico-thyroid muscle, while the inferior or recurrent laryngeal supplies all the other muscles that act on the glottis, and contains the sensory fibres for the larynx below the vocal cords. The vocal cords are abducted, and the glottis opened, chiefly by one muscle—the posterior crico-thyroid—which passes upwards and outwards from the back of the thyroid to the outer muscular process of the arytenoid; and, drawing this back, moves the vocal process outwards. The cords are adducted, and the glottis closed, by several muscles, of which the most important is the lateral crico-arytenoid, which, passing backwards from the side of the cricoid cartilage to the outer process of the arytenoid, moves this forwards, and is thus the opponent of the posterior muscle. But the outer fibres of the thyroid-arytenoid muscle, which pass, parallel to the vocal cord, from the thyroid cartilage to the muscular process of the arytenoid, have a similar although feebler action. This closing rotation of the arytenoids is supplemented by the arytenoideus, which, passing from the back of the one to the back of the other arytenoid cartilage, brings the two together. The vocal cords are lengthened and made tense by the criĉo-thyroid, which draws back and slightly tilts the cricoid cartilage, and they are made tense or lax in parts by the inner fibres of the thyro-arytenoideus, which end at different points along the cord. But the muscular mechanism must be much less simple than this. In the case of some muscles, all the fibres have not the same direction, and may have different actions according to their association. Doubtless, in the delicate and varied actions that produce vocal sounds, complex associated actions of the fibres are concerned.

The symptoms of paralysis are threefold—altered phonation, deranged regulation of the entrance of air in breathing, and defective movement (observable with the laryngoscope). The phonic and respiratory functions of the glottis are subserved by the same muscles and the same nerves, but by centres that must differ in their anatomical connection, if they do not in their anatomical position. After death, the vocal cords are in a position of slight abduction from the middle line, and this, termed the "cadaveric position," must be regarded as that of muscular relaxation—of that rest which, during life, they never actually attain, since they move with every breath. They move farther apart during inspiration; they come nearer together during expiration; while in phonation they are brought very near together and made more tense. In total paralysis of all the muscles, the cords are in the cadaveric position, and do not move with breathing or on an attempt to produce sounds. Instead of the natural explosive cough there is only a rush of air through the glottis. There is some stridor on a forcible inspiration. If only one vocal cord is completely paralyzed, some hoarse phonation may still be possible by extreme adduction of the normal cord, and its abduction prevents inspiratory stridor, but a proper cough is still impossible. Such one-sided palsy may result from disease of the nucleus of the spinal accessory in the medulla, of its root at the surface of the medulla, of the trunk of the vagus, and even of the recurrent laryngeal, since the escape of the crico-thyroid in the latter case does not materially modify the symptoms. Hence the palsy itself does not help us to determine the position of its cause.

In other cases of bilateral palsy the cords are nearer together than the cadaveric position. They can be approximated for voice or cough, and when the effort is over, their elasticity may make them recede a little; but they cannot be abducted even as far as the cadaveric posture, and the normal separation during inspiration does not occur. This is called paralysis of the abductors, the posterior crico-arytenoids. The longer the palsy lasts, the closer together are the cords, in consequence of a secondary contracture of the unopposed adductors. The cords being always in the position of phonation, voice is little affected, but the absence of the normal separation during inspiration causes a serious impediment to respiration, since the inrushing air brings the cords still nearer together, and causes a loud inspiratory stridor, and dyspnœa on the least exertion. The absence of expiratory stridor distinguishes the dyspnœa from that due to tracheal stenosis, and, together with the integrity of the voice, distinguishes this from any other laryngeal affection. The condition is one of considerable danger, since the least catarrhal swelling of the mucous membrane may necessitate immediate tracheotomy to prevent suffocation. If this abductor palsy is one-sided, the symptoms are slight or absent, and the diagnosis can be made only by the laryngoscope. The most frequent cause of abductor palsy is central degeneration; but it sometimes occurs, strange to say, in severe hysteria, even, as I have seen, in extreme degree. Equally strange is the well-established fact that it may result from disease of the recurrent laryngeal, which, supplying the adductors also, should cause only a total palsy. This has given rise to much speculation as to its mechanism. Some light seems to be thrown on it by the fact that electrical stimulation of the recurrent laryngeal also causes adduction, although all the muscles, adductors and abductors, must be equally stimulated to over-action. This must be due to the greater power of the adductors, perhaps also to the mechanical advantage at which the chief adductor, the lateral crico-arytenoid, acts, in comparison with the abductor, the posterior crico-arytenoid; since the former passes nearly at right angles, the latter at a very acute angle, to the muscular process of the arytenoid cartilage. A force acts on a lever at greatest advantage when applied at right angles to the lever. The effect of a general under-action may be to impair the effect of the abductors more than than that of the adductors, just as the general over-action on electrical stimulation increases the effect of the adductors out of proportion to that of the abductors. According to this explanation, the abductor palsy would be the effect of paralysis of the recurrent, when it is incomplete in degree, although not necessarily partial in distribution; while the total palsy with cadaveric posture would be the result of complete paralysis. In harmony with this is the fact that in progressive disease of the recurrent an initial abductor palsy has been observed to pass into total palsy (Schech, Rosenbach). In some cases, as Riegel suggests, secondary contracture of the crico-thyroid may aid in causing the adduction; but since adduction is often absent in total recurrent palsy, the influence of this contracture cannot be great. When the palsy is first, and for long, of the abductor type, secondary tissue-changes in the preponderating adductors may perhaps maintain the glottis in adduction, even when the palsy has become complete.

In simple adductor palsy the cords are apart, and cannot be brought together, but further abduction occurs in deep inspiration. The cords are not approximated in speech, and so the patient is voiceless, but they can still be brought together in coughing. Hence it has been termed by Türck, "phonic paralysis."[1] This is the cause of hysterical aphonia, but is scarcely ever produced by organic disease. It is readily distinguished, by absolute voicelessness and perfect cough, not only from other laryngeal palsies, but also from other diseases, such as catarrhal laryngitis, in which there is a hoarse attempt at phonation.

These laryngeal palsies can be diagnosed with certainty only by the aid of the laryngoscope; but the symptoms themselves often justify a strong suspicion of the nature of the affection, and it may be well to put them before you in the form of a table. You will see that the inability to effect an explosive cough is of great significance, and should lead you to suspect a palsy, probably of organic origin; you will observe that if voice is also entirely lost, the palsy must be double. If voice is preserved and cough lost, you suspect one-sided palsy. Loud inspiratory stridor with preserved voice means double abductor palsy; a normal cough and no voice or stridor signifies an unimportant adductor palsy.

SYMPTOMS.	SIGNS.	LESION.
No voice; no cough; stridor only on deep inspiration.	Both cords moderately abducted and motionless.	Total bilateral palsy.
Voice low-pitched and hoarse; no cough; stridor absent or slight on deep breathing.	One cord moderately abducted and motionless, the other moving freely, and even beyond the middle line in phonation.	Total unilateral palsy.
Voice little changed; cough normal; inspiration difficult and long, with loud stridor.	Both cords near together, and during inspiration not separated, but even drawn nearer together.	Total abductor palsy.
Symptoms inconclusive; little affection of voice or cough.	One cord near the middle line not moving during inspiration, the other normal.	Unilateral abductor palsy
No voice; perfect cough; no stridor or dyspnœa.	Cords normal in position and moving normally in respiration, but not brought together on an attempt at phonation.	Adductor palsy.

The nerve-supply to the palate is one of those points in anatomy that urgently require re-investigation. The most important muscle is the levator palati, which receives a nerve from the spheno-palatine ganglion, but the ultimate origin of its fibres is certainly not from the fifth nerve. They are usually said to come by the Vidian from the facial, but they probably really come from one of the bulbar nerves, either the glosso-pharyngeal or spinal accessory. Paralysis of the palate results from disease of these bulbar nerves (at the surface of the medulla) or of the bulbar nuclei. It is doubtful whether it ever results from disease of the facial nerve, and certainly it does not from disease of the fifth. The centres for the palatine muscles are especially obnoxious to the mysterious influence that diphtheria leaves behind it, and this is the most frequent cause of simple palsy of the palate. In total palsy, the soft palate hangs low and flaccid, the uvula is long, and no movement occurs in drawing a deep breath, in phonation, or on tickling the mucous membrane. The posterior nares are no longer closed. Hence during swallowing, liquids are apt to come back through the nose; during speaking there is a nasal resonance, and the explosive consonants, as *p* and *b*, are no longer properly articulated, because the patency of the nares prevents the necessary compression of the air in the mouth, and they are transformed into *m*. In unilateral palsy these symptoms are absent. All the muscles, even of one side, are rarely paralyzed, since unilateral palsy is scarcely ever due to disease of the centre (which affects both sides), and the course of the fibres to the different muscles is not the same, and therefore a lesion does not affect all of them. The palate at rest presents little change in unilateral paralysis. On one side it may be a little lower than the other. The uvula may hang a little to one side, but palsy never causes any curve in the uvula, and no abnormal position of the palate or uvula can be due to paralysis, unless it is increased on movement. The most important symptom of one-sided palsy is recognized only on movement. At rest, the palate may appear equal, but if the patient is made to say "ah," the base of the uvula deviates a little to the unaffected side, and a little on this side of the middle line, about midway between the arch and the hard palate, a slight dimple forms, while the unparalyzed side remains smooth. The difference is obvious and characteristic. There is no deviation of the uvula. The difference between the two sides, on movement, evidently depends on paralysis of the levator palati. It occurs together with paralysis of the tongue and vocal cord on the same side, when there is disease at the side of the medulla damaging the hypoglossal and spinal accessory nerves. This was first pointed out by Dr. Hughlings-Jackson.

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[1] A much more exact term than his attractive but loose antithetic designation of "respiratory paralysis" for abductor palsy.

Whether the paralysis depends on the disease of the spinal accessory, or on disease of the adjacent glosso-pharyngeal, is uncertain, but the association leaves no doubt that the motor fibres for this, the most important of the palatine muscles, come from one of these bulbar nerves. I have known the same three palsies (tongue, palate, and vocal cord) to result from a deep-seated tumor in the upper part of the neck, which must have damaged the nerves outside the skull.

When the accessory part of the spinal accessory is injured by disease at the foramen magnum, the spinal portion may suffer also, causing wasting and loss of power of the sterno-mastoid and upper part of the trapezius.

Disease of the hypoglossal nerve, outside or within the medulla, causes paralysis of the same half of the tongue; and disease of the motor tract above the nucleus causes paralysis of the opposite half of the tongue. Disease of the nucleus itself almost always causes paralysis of both sides of the tongue, because degeneration affects both nuclei, and the two lie so near together that both suffer in acute lesions. In one-sided palsy, the tongue, at rest, is in its normal position in the mouth, but the base is higher on the paralyzed side, owing to lack of tone in the posterior fibres of the hyoglossus. Within the mouth, movement is deficient towards the paralyzed side, but on protrusion the tongue deviates from the sound, and towards the paralyzed side, being pushed out and over by the unaffected genio-hyoglossus. In bilateral palsy the tongue lies motionless within the mouth, and cannot be moved. In disease of the nerve or nucleus, the tongue usually wastes, and the mucous membrane lies in irregular folds over its surface.

The process of articulation is effected by the muscles supplied by these bulbar nerves, and its impairment always constitutes an important symptom of their disease, or of disease in the part of the brain from which they arise, and in which a complex structural association subserves the conjoined action of the various muscles concerned in the process. Articulation consists in stopping and varying the outgoing current of air, which is often thrown into vocal vibrations, on which the process of articulation effects more elaborate and perfect modulations than it can in merely whispered utterance. In the whole series of movements that can be effected by the muscles of man, there are none comparable to these in exquisite delicacy of adjustment, and in the infinite diversity of result that a few simple muscles can produce by their combined action. It is not surprising that commencing failure of the adjustment of nervous action should be manifested in these sooner and more than in any other mechanism. There are two chief forms of defect of articulation. The first is due to paralysis of the muscles, and in this the loss of power can be recognized in other movements. The precise form of defect depends on the muscles that are weak, and this I have already alluded to, and will return to in a moment in speaking of the combined palsy. In the other variety there is no actual paralysis, but the delicate adjustment is at fault. Syllables are run together, in what may be termed "confluence of articulation;" the ends of words are not well pronounced, are even elided; or the syllables may be unduly separated, in what is termed, from a musical analogy, "staccato" utterance. The most delicate of all the movements is that involved in the pronunciation of *r*, and it is in this that the commencing defect is often most conspicuous, as by making the patient say "truly rural." Often there is a peculiar drawl, and this may be combined with elision of syllables. In many cases, and especially in the paralytic defect, the patient can utter a word by a deliberate effort much better than he does in habitual speech.

Before leaving the subject of the symptoms of the cranial nerves, one other subject should be noticed. Certain of these nerves are liable to suffer together from disease of their nuclei, usually from degenerative disease. The nerves thus associated in disease are the two groups of motor nerves that are associated in function. The first group is that of the nerves for the eyeball muscles, and its disease causes the progressive ophthalmoplegia that I have already mentioned. The other group is that of the nerves for the complex series of muscles of the orifice and upper part of the respiratory passages, mouth, throat, and larynx—the fibres of the facial for the orbicularis oris, the hypoglossal, the fibres to the palate, perhaps from the glosso-pharyngeal, those that give motor power to the pharynx, and the laryngeal fibres of the spinal accessory. Before considering the associated palsy that results from nuclear degeneration, I may remind you of the associated one-sided palsy of tongue, palate, and larynx, which I have just described as the result of disease outside the medulla. The tongue deviates to one side; the middle of the soft palate is drawn to one side when it is raised, and if the larynx is examined, the vocal cord on that side is seen to be motionless. These symptoms are due to damage to the roots of the hypoglossal, spinal accessory, and perhaps also of the glosso-pharyngeal.

In the associated nuclear palsy—"bulbar paralysis," as it is often termed—the paralysis involves the same parts, tongue, palate, and vocal cord; but it is bilateral, and its functional distribution is more complete, since the lips and pharynx are usually also involved. You will remember what I said of the central relations of the nerve-fibres for the lips: although they run in the facial nerve, they must be derived from nerve-cells that have the most intimate connection with those for the transverse muscle of the tongue, and the two may even be derived from the same nucleus. The association of the lips with the other parts caused Duchenne to give to the disease the name labio-glosso-pharyngeal paralysis," by which it is still often known. The symptoms are, as it were, grouped about the tongue as a centre, and it is in the delicate movements of lingual articulation that the first symptoms occur—a clumsiness in the pronunciation of the lingual consonants *l, r, n, t,* and *s.* Subsequently the degree of protrusion of the tongue becomes impaired, until at last only the tip can be put beyond the teeth. It is often conspicuously wasted. The early weakness of the lips prevents whistling, and the labial explosives *b* and *p* become *f,* and *v* becomes *u.* This transformation is assisted by the weakness of the palate, which ceases to shut off the nasal cavity, and so interferes with the compression necessary for explosive sounds; hence also nasal resonance persists in all sounds. Swallowing becomes difficult; liquids regurgitate into the nose; food gets into the larynx, and this may be so paralyzed that the glottis cannot be closed, and an explosive cough is impossible. Ultimately a low, hoarse vocal sound is all that remains of speech; but it is rare for the paralysis of the larynx to be complete.

LECTURE IX.

SYMPTOMS (*Continued*): MENTAL DISTURBANCE—LOSS OF CONSCIOUSNESS—APOPLEXY—DELIRIUM—MENTAL WEAKNESS—LOSS OF MEMORY.

GENTLEMEN,—The symptoms that have hitherto engaged our attention are the derangements of special functions subserved by special nervous structures. Those that we have now to consider are more general in nature, and the symptoms caused by their derangement are for the most part "diffuse." It is convenient to consider with these some symptoms that are "focal," but which are closely allied to those that are general. Thus it is better to study defect of speech, a focal symptom, after we have considered derangement of mind, a general symptom.

The highest functions of the brain are those concerned in mental processes, and the derangement of these is a frequent and obtrusive effect of organic brain-disease. But these symptoms do not stand in the special and pre-eminent relation to such disease that might reasonably be anticipated. The highest cerebral functions are so readily disturbed, that their derangement is less frequently the result of organic brain-disease than of changes in the blood, on the one hand, or of the minute alterations of nutrition that we term functional disease, on the other hand. The most significant disturbance from organic disease is the coarsest, such as unconsciousness. The slighter disturbance, such as delirium, derives its significance from its associations, not from its simple presence.

We may consider first the profound and important mental symptom that I have just mentioned—loss of consciousness. Remember that the terms "conscious" and "consciousness" are used in two senses: first, to signify subjective knowledge of the occurrence of mental processes; and secondly, outward manifestation of such processes. In medical language the words are chiefly used in the second of these two senses. A patient is said to be "unconscious," or to have "lost consciousness," when there is no evidence of mental action, either spontaneous or in response to attempts to elicit it. The term "insensible" is often applied to the same condition. Another confusion is introduced by the use of the term "conscious of," or "unconscious of," in the sense of cognition, or its absence. Thus a delirious patient is said to be unconscious of what is occurring around him, although he is not said to be unconscious.

Loss of consciousness may occur suddenly or gradually, may be complete or incomplete. The variations may be in the degree of subjective consciousness, or of the external manifestation of consciousness; and it is to the latter that the term "partial loss" is usually applied, as, for instance, to the condition in which a patient lies apparently asleep, but opens his eyes when spoken to, immediately relapsing into sleep. This condition is often termed "stupor." Complete unconsciousness, lasting more than a few minutes, is termed "coma." In both conditions there is usually imperfect control over the sphincters. In stupor, the reflex action on the limbs is preserved, and sometimes increased; the patient swallows automatically liquid placed in his mouth; the pupils act to light. In coma, the reflex action in the limbs is usually lessened and often lost. Muscular tone gives place to flaccidity, and with this change myotatic irritability often disappears. The pupils may be widely dilated or small, and do not act to light, at any rate when the coma is deep, and then the conjunctival reflex is also lost. The act of swallowing may or may not be possible; in deep coma it is lost, and the palate, sharing the muscular relaxation, vibrates under the current of air, and causes the peculiar "stertor" which is a familiar indication of the depth of coma. Even the respiratory movements are lessened, in consequence of lowered activity of the respiratory centre; they become shallow, infrequent, and sometimes present rhythmical variations of intensity, in what is termed the "Cheyne-Stokes breathing"—alternating periods of decreasing and increasing depth of breathing, separated by a pause. The lessened breathing fails to clear the air-passages of the secretion in them; this accumulates in the bronchial tubes, and is often erroneously regarded as evidence of bronchitis; finally, mucus collects in the trachea, and causes the well-known harbinger of death.

Consciousness may be impaired by almost any one of the many morbid processes to which the brain is liable, whether acute or chronic. It results from chronic and subacute disease chiefly when this damages a considerable area of the cortex, either directly, or indirectly by causing a rapid or extreme increase in the intracranial pressure. It results from sudden lesions in any part of the brain, and is then usually sudden in onset, and termed "apoplexy."

Of all the sudden lesions that cause apoplexy, intracranial hæmorrhage is the most effective, and the most frequent; hence "apoplexy" has come to be a synonym for internal hæmorrhage, whatever be its seat. Next in frequency is the sudden occlusion of a large artery by a plug brought from a distance (embolism), or formed *in situ* (thrombosis). It may result from congestion of the brain, although it does so far less frequently than is commonly supposed. A similar sudden loss of consciousness may occur in the old without any visible lesion of the brain to which it can be ascribed. This has been termed "simple apoplexy." In senility the brain shrinks; the space between the convolutions is occupied by serum. This change, which is common in the old, and without significance, when found in old persons who had died of simple apoplexy, was thought to be important, and the cause of death; hence the condition was termed "serous apoplexy"—a disease that has no real existence, although the name still survives, and now and then finds its way into certificates of death.

The characteristic of apoplexy is sudden loss of consciousness, not due to any cause outside the nervous system, such, for instance, as failure of the heart's action or a poison in the blood. The onset may be sudden; the patient falls as if "struck" down by some unseen hand,—an idea that is fossilized in the name, and in its English synonym, a "stroke." Sometimes the onset is gradual; consciousness slowly fades; stupor slowly deepens into coma: and this has been termed "ingravescent apoplexy." The face may be flushed or pale; it is rarely very pale. Often the heart and arteries pulsate strongly, but sometimes less frequently than normal. The temperature is usually at first depressed; its subsequent course varies with the cause of the apoplexy. An exception to the initial depression of temperature is presented by active lesions of the pons, which, whatever be their nature, are sometimes attended with a rapid rise of temperature, that may, in an hour, reach 105° to 106°. If the attack is one of moderate severity, reflex action soon returns, and in the course of a few hours some indication of returning consciousness can be perceived. On the other hand, the coma may deepen, and the interference with breathing, already described, may come on. In most cases the symptoms of apoplexy are accompanied by those of a local cerebral lesion, commonly by those of hemiplegia, previously detailed.

The symptoms of apoplexy are those of lowered cerebral function, beginning at the highest, and extending downwards to lower centres in proportion to the depth of the coma. Its precise mechanism has been much discussed, but is a matter of theoretical rather than of practical importance. It is easy to frame a simple and satisfactory hypothesis of the way it is produced by any one lesion, but the variety of its causes shows that more than one mechanism may be concerned in its production, and suggests that its origin is complex in every case. Sudden increase of intracranial pressure causes loss of consciousness, but in what degree the result is due to the mechanical action on the nerve-elements, or to anæmia from the compression of the capillaries, is uncertain. This mechanism is doubtless effective in cerebral hæmorrhage, but it is probably not the sole mechanism, even in this case, because apoplexy may result from a very small hæmorrhage, and consciousness may be lost at the very onset of a hæmorrhage. The sudden occlusion of a vessel will cause apoplexy, but cannot do so by its influence on the intracranial pressure. Attempts that have been made thus to explain it are remarkable chiefly for their ingenuity. In most cases of apoplexy there is a sudden damage to the cerebral tissue. Sudden arrest of blood-supply constitutes a damage as effectual as laceration. Hence it is probable that one element in the production of the loss of consciousness is the inhibitory effect of the irritation of the lesion. We have evidence of a downward influence of this character in the initial loss of muscular tone and muscle reflex action (*e.g.*, in the loss of the knee-jerk[1]). A similar upward action, inhibiting the highest centres, is probably the cause of the initial loss of consciousness, the prolongation of which may be helped by other mechanisms, such as increased pressure. All lesions are effective in proportion to the rapidity with which they are produced. Experiments, for instance, show that the amount of intracranial pressure needed to abolish consciousness has to be ten times greater when slowly than when rapidly produced.

The diagnosis of the cause of apoplexy we shall consider when we come to the last part of our subject—to the indications of the nature of the brain disease,—but I may briefly point out to you some of the chief differences between the coma of cerebral origin and that which may result from causes outside of the nervous system, or from cerebral derangement that is merely functional in character. In all cases the most important point is to search carefully for any evidence of a local cerebral lesion, and especially for the indications by which hemiplegia can be recognized during the state of coma. These I have already described to you (p. 46). The reflex actions are of especial importance. If these are all perfectly normal, this is, in the absence of other decided symptoms, against the existence of a cerebral lesion. An abnormal condition of reflex action is in favor of it, and is almost conclusive if the abnormality is unilateral.

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[1] See "Diagnosis of Diseases of the Spinal Cord," 3d Ed., p. 31.

We will take first the functional disorders of the nervous system. A patient may be unconscious for an hour or two after an epileptic fit, and this may be mistaken for apoplexy with a convulsion at the onset. Such a difficulty can scarcely arise unless the history of the patient is unknown, since first convulsions are rare, except in those in whom cerebral apoplexy is also rare. Post-epileptic unconsciousness resembles sleep more than it resembles coma. The patient can be readily roused. The temperature is nearly normal—never below normal, as it often is in apoplexy,—and unilateral symptoms are absent. The convulsions that cause transient post-epileptic hemiplegia are always one-sided, and are not followed by such deep sleep as to raise the question of a possible cerebral lesion. The patient soon emerges from the mental obscurity that follows an epileptic fit, and often at once passes into a normal condition, but occasionally is still "befogged" for a longer or shorter time—in a wandering, stupid state of mind, that is itself very characteristic.

The state of unconsciousness that occurs in rare cases of hysteria simulates apoplexy less than it resembles the coma due to less rapid cerebral processes, such as meningitis. A patient, for instance, after a period of headache, becomes unconscious, swallows what is put in the mouth, but cannot be roused. The diagnostic indications are the age and sex of the patient, who is usually either a female or a boy; the absence of any cause of a cerebral lesion; the history of other symptoms of functional nervous disturbance (especially of hysterical convulsions or vertical headache); the cessation of headache when the coma comes on; the absence of all objective symptoms; the inconsistence between the ready deglutition and the apparent depth of coma; and the uniform course of the affection, which often presents no change for many days.

Of causes outside the nervous system, uræmic poisoning is that which most often causes perplexity, because its frequent cause, chronic kidney-disease in the second half of life, is also a frequent cause of the apoplexy that it resembles. Albuminuria is constant in uræmia, frequent in apoplexy; its absence is therefore more significant than its presence. In many cases the coma is preceded by other uræmic symptoms, especially by convulsions or amaurosis. Severe general convulsions may, it is true, usher in both affections, but at the onset of apoplexy the convulsion is usually single; at that of uræmic coma there are many. Sudden complete amaurosis is almost conclusive evidence of uræmia. An acute cerebral lesion scarcely ever causes total blindness. Elevation of temperature is strongly in favor of cerebral mischief; depression is consistent with either, but continuous depression, lasting for two or three days, is strongly in favor of uræmia. An examination of the fundus of the eye will, of course, be one of the first steps you take. Albuminuric retinitis often proves the existence of renal disease, when an examination of the urine is, for the time, impracticable. It shows, moreover, profound systemic mischief. It always indicates that the patient is in a state in which uræmic symptoms may come on at any moment. The absence of retinal change does not exclude uræmia, any more than its presence proves that uræmia is the cause of the coma.

In profound alcoholic poisoning the diagnosis from cerebral apoplexy may be extremely difficult. When you have no history to guide you, as is often the case with the patients who are brought to a hospital or a police-station, the diagnosis may be impossible. Many a patient with apoplexy has been locked up in a police-cell all night as dead-drunk. Brandy is the universal panacea for impairment of consciousness; and so apoplectic patients often smell of alcohol. If focal symptoms are absent there is no distinctive indication. A few hours will always decide the question; and it is better to let a drunken man get sober in bed, than to let a patient with ventricular hæmorrhage die in a police-cell. We need, however, more facts regarding the state of reflex action in profound alcoholic poisoning, especially of the muscle reflex action (knee-jerk, foot-clonus, etc.). It is not improbable that some useful guidance may be found in these symptoms. This is one of the many points on which those of you who are hospital residents may make valuable observations.

The danger of confusing opium-poisoning and apoplexy is not great, because the patient who has taken opium usually comes under observation while the pupils are contracted to an extreme degree—conclusive, except as regards the distinction from hæmorrhage into the pons. In the latter there is often a history of sudden onset, and usually objective symptoms are present that permit no doubt as to the nature of the case.

From the lessened manifestation of mental activity we may pass to the disorder and excess that constitute "delirium," the condition in which mental processes are not in accordance with sense-impressions, and there is no consciousness of the discrepancy. The condition is essentially the same as that which constitutes "insanity;" but the term delirium is used when the mental derangement is acute in course, and occurs in consequence of organic brain disease or of some blood-state. Delirium is commonly distinguished as "quiet" or "active." In the former there are hallucinations, especially of sight, and these dominate the patient's ideas. He often talks continuously, but in a low monotonous voice, and it may be difficult to make out what is said—a condition aptly termed "low muttering delirium." On the other hand, in "active delirium" there is more energy in the manifestation of the mental processes, and the patient tries to act according to his erroneous ideas. Although the elements of delirium are identical in nature with those of what is termed insanity, certain common features of the latter rarely occur in delirium. Such are the extreme and persistent emotional depression of melancholia, the exaggeration of idea that is common in general paralysis of the insane, the outrageous delusions of personal identity met with in some cases of chronic insanity, and the rhetorical loquacity of acute mania.

Delirium is far less frequently the result of organic disease of the brain than of altered conditions of the blood, especially that in pyrexia. There is rarely anything in the character of the delirium to indicate to what cause it is due. In acute alcoholic delirium, unpleasant visual hallucinations are a marked feature, and there is usually conspicuous tremor; but chronic alcoholic delirium may present none of these characteristics. The delirium due to pyrexia and that resulting from organic brain disease present no difference. Since pyrexia is by far the most common cause of delirium, this symptom alone is suggestive of organic brain-disease only when there is no pyrexia, or only a slight degree of pyrexia, insufficient to account for it. It must, moreover, be remembered that those who are addicted to alcohol, and those who are old, are rendered delirious by a slighter degree of blood-change than is necessary in the case of the sober and the non-senile. The caution to attribute no weight to delirium, unaccompanied by other symptoms of brain disease, if the patient has considerable fever, may seem a simple rule; but, like many other simple rules in diagnosis, it is often forgotten, and consequent mistakes are frequent. Not long ago I saw a man who was said to have inflammation of the brain, and I found he had only inflammation of the lungs. The delirium had so misled the doctor in attendance that he had not even examined the lungs. Again, a child became feverish and delirious: the medical attendant diagnosed tubercular meningitis, and foretold a speedy death. But the pyrexia was sufficient in degree to account for the delirium: there was no evidence of anything more than a catarrhal febricula, and in a week the child was well. Do not, however, go to the other extreme, as some have done, and conceive that delirium is of no significance as an indication of organic disease. It is significant when there is no other discoverable cause, and it may both confer and receive significance by association with other cerebral symptoms. These may be various in character, but one of the most frequent is headache. Headache, like delirium, is an effect of fever. But the headache of fever ceases when the delirium comes on; that of brain disease persists. The coexistence and the sequence of the two have, therefore, quite a different significance. If the patient is delirious, and has, at the same time, severe pain in the head, you should suspect organic disease. Of course, pyrexia often accompanies delirium from organic disease, and we have then to depend on the other symptoms for our diagnosis, or on the order of the two. If the delirium precedes the fever, it has evidently the same significance as if it existed alone. The various other symptoms that may give significance to delirium need not be enumerated here.

Mental weakness shows itself in failure of power in all the various mental processes, and of these loss of memory ("amnesia") is the most conspicuous and the most tangible. Memory, like other mental actions, has its physical side. Every functional state of the nerve-elements leaves behind it a change in their nutrition, a residual state, in consequence of which the same functional action occurs more readily than before; and this residual disposition is increased by repetition. This is the basis of motor training, which consists in a sort of motor memory that enters little into the region of consciousness. The same residual disposition in the cells that act during mental processes no doubt influences the revival of those processes in memory properly so-called. The sequence of action of groups of nerve-cells is the physiological aspect of that which, in its psychological aspect, we term the association of ideas. There is no special faculty of memory, physical or psychical, apart from the general cerebral and intellectual processes. But there is, or seems to us to be, a peculiar power of the voluntary revival of these processes —of the re-energizing of the residual tendencies,—a faculty that is popularly termed "recollection."

Any disease of the brain may affect memory, whether it be coarse organic disease or finer degenerative processes. Moreover, temporary malnutrition, as from acute disease, or severe anæmia, may have the same effect. The defect may be seen in the inability to retain new impressions, or in the loss of those that are recent. As Ribot has put it, "the new perishes, the old endures." Strange examples of this are sometimes met with. I have seen a clergyman who had lost all memory of the last twenty years of his life. Those years had passed over him, leaving their marks indelibly on his frame. They had been years of active work, and at the end of them he had an illness. When he recovered, all memory of those years had vanished. In rare cases of the kind, memory has returned, and the lost time comes back in the order from the past towards the present.

Another symptom of mental failure, with which, indeed, loss of memory is closely associated, is deficient power of attention, of excluding all but one subject from the domain of consciousness. This may be one cause of failure of memory, and it sometimes causes a failure to remember when there is no real failure of memory. When one subject dominates the mind, sufficient attention is not given to other subjects to secure their retention. Hypochondriacal patients often present this inability; their minds are constantly occupied with their own feelings, and they do not give sufficient attention to other subjects to insure their persistence in the mind, and the apprehension of mental failure is added to the other sources of mental distress. Reassure the patient as to his fancied ailments, and the unmeaning character of his various sensations, and his loss of memory will vanish.

Closely connected with failure of the power of attention is incoherence of idea. Instead of the definite sequence of mental processes that we recognize as normal, one mental image excities another by some accidental association, which would be unnoticed in health. Often the connections that determine sequence are so subtle as to evade detection. The mental processes change rapidly, and when one is only half expressed, another has possession of the mind. This incoherence is conspicuous in delirium, and is also frequent in simple mental failure.

Defect of moral sense is also common in mental failure. The slighter defects are relative, rather than absolute, and must be estimated by their deviation from the normal conduct of the individual. Many actions would be more distinctly pathological in a man of refinement than in an ill-mannered man of the lower classes. Other actions are unequivocal. Urine and stools are often passed into the bed in cases of brain disease, in consequence of this mental failure, when there is no loss of power over the sphincters. Under these circumstances it is an indication of a considerable degree of mental change—a greater degree than is suggested, perhaps, by the other indications of the mental state. Of similar significance, in these cases, is a disinclination to swallow. There is no real difficulty in deglutition, but when food is placed in the mouth, the patient lets it lie there, and after some time, perhaps half an hour, spits it out again. Particles may get into the larynx, and suggest that there is a pharyngeal paralysis that does not really exist. It is important to note that, in children, slight mental defect is often shown by a lack of the sense of propriety, rather than by failure of mere intellectual processes. They are unabashed by the presence of strangers, are disobedient, mischievous, meddlesome. Indeed, the lack of capacity for restraint often leads to undue manifestation of what mental power they possess, and parents constantly consider such children unduly precocious, and possessed of mental faculties above the average.

LECTURE X.

SYMPTOMS (*Continued*): AFFECTIONS OF SPEECH.

GENTLEMEN,—From the mental symptoms that we considered in the last lecture, we pass now to another group, intermediate between the mental symptoms on the one hand, and simple motor and sensory symptoms on the other—affections of speech. We must consider them at some length, because the phenomena are complex, their relations are intricate, and if I were to attempt to be brief, I should succeed only in being unintelligible. As it is, I fear I must ask for your somewhat close attention. There is difficulty, not only in the theoretical study of the subject, but also in the practical application of our knowledge. No two cases of speech-defect are alike; and you can only unravel the phenomena of each case by having a firm grasp of the laws that govern both normal speech and the derangement that is produced by disease.

The brain contains upper and lower mechanisms for expression by articulate speech, the upper in the cortex, the lower in the medulla. The latter transfers to the peripheral nerves the impulses that come down from the cortex, perhaps adjusting their form in minor details. It is in the cortex that the elements of speech are arranged. In disease of the lower mechanism, the elements of expression are correct in nature, number, and arrangement, but their form is defective; "articulation" is at fault—the jointing of the elements. In disease of the higher cerebral apparatus, the form of the constituent elements may be correct, but they are wrong in nature, in number, or in arrangement. The error in the arrangement of the elements often causes great error in the form of words, although the form of the elements may be correct. You will understand this better as we proceed. We have already considered the effect of disease of the lower mechanism in the medulla, and are now concerned only with the cortical mechanism.

Speech is the expression of mental processes; but it is not the only mode of their expression. They may also be expressed by writing; but writing is merely expression by speech translated into symbols of a different kind. The nervous processes are elaborated in the same cortical mechanism, although they leave the cortex at a different place, and do not pass through the lower mechanism for articulate speech, but pass by it, to still lower mechanisms in the spinal cord.

A different and very important mode of the expression of mental processes is by simple muscular movements in various parts of the body—face, limbs, and even trunk; we call these "gestures." Gesture-symbols are much simpler than speech-symbols. They are the first to be acquired by the race and by the child—the most uniform in different races. Speech-symbols are acquired later, and are diverse in different races.

By these methods we express two classes of mental processes—"ideas" (which are expressed as propositions) and emotions. Emotional processes are by far the simpler, and they are expressed by the more simple and automatic methods, chiefly by gesture. This is the chief use of gesture. It is true, gesture can express propositions, but only in a very limited degree, and only those that are extremely simple in character. An instance is the expression of affirmation by a nod. Gestures that express propositions are called "signs."

In vocal speech there are two elements—articulation and phonation. Articulation forms the words on the outgoing current of air; the larynx adds voice to speech, and enables it to be heard at a distance. Voice is merely material for articulation. It is indeed a means of expression, by its variations, but it is only an expressional gesture accompanying speech, and, like other gestures, it expresses chiefly emotions. Emotions may also be expressed by words, as by interjections, and by many phrases that have a propositional form, but only an interjectional meaning—"dead propositions," they have been aptly termed by Hughlings-Jackson, to whose philosophical investigation of aphasia every student of the subject is profoundly indebted.[1] Most oaths are such "dead propositions," and so are many familiar expressions, as when, to express mere surprise, we formally deny, as in the phrase, "You don't say so!" Real propositions, as such, cannot *express* emotion, they can only state the fact of its existence. Although tone chiefly expresses emotion, it has, like other gestures, a limited power of conveying propositions, as when a negation is converted into a question by an interrogative tone.

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[1] Of Dr. Hughlings-Jackson's writings on the subject, the most important are his papers in "Brain" (vol. i., p. 305; ii., p. 203, 323). The student who is interested in the subject is strongly advised to read these articles. Those who are acquainted with them will see how largely the views expressed in this lecture have been moulded by those of Dr. Jackson, and how extensively I have adopted the phraseology that he has made, not only current, but indispensable.

The expression of emotion is essentially involuntary and automatic. The will is needed, not to effect it, but to restrain it. The expression of propositions is chiefly voluntary, by an effort of the will. But propositions differ much in their speciality. The more special, the less frequently expressed they are—the greater is the volition required, although we may be scarcely conscious of it. The less special, the more frequently employed they are—the less voluntary, the more automatic is the utterance. In vocal music, words are chiefly used as the vehicles for tone. The propositions that the words formally convey are scarcely ever really expressed as such.

Intellectual processes are aroused by language as well as expressed by language—aroused through the senses of hearing and of sight, and in blind persons by touch. This necessarily involves an intimate connection between the nervous processes for these sensations and those for language. Of the links of this connection we can recognize some, because disease occasionally separates them. When a word is heard, the processes thus aroused in the auditory centre excite others that subserve the recognition of the sound as a word, and these, in their turn, excite those that subserve the image corresponding to the word. So too with visual word-symbols. Thus the nervous processes for language have both motor and sensory relations. Of these, the sensory (auditory) processes are developed earlier than the motor processes. The child understands many things said to him long before he can utter a single word.

We have seen that a sensory word-process must intervene between that for the sound and that for the image of the thing symbolized. So motor word-processes intervene between those for the image and the motor impulses for the muscles. There is a sort of internal revival of words before they are uttered, and this may occur without utterance. A revival of word-processes, motor or sensory, often accompanies deliberate thought.

The motor and sensory word-processes leave behind them residual states, which facilitate the subsequent revival of the same arrangements. These residual states subserve the memory for words. We have seen that memory of other kinds is subserved by similar residual states (p. 101). Thus there may be said to be both a motor and a sensory memory for words. In the revival that precedes speech, we are more conscious of the motor process, of a sort of "internal speech." But it is probable that both motor and sensory word-processes occur together, and that the sensory (auditory) word-process actually leads in the revival. This process, as we have seen, is first established. It may be perfect when the motor memory is lost, and its loss interferes with thought far more than does the loss of the motor memory. In the act of reading words, the process is analogous to that which takes place in hearing spoken words. Nerve-processes are aroused successively for the simple sensation, the word-symbol, and the image of the thing symbolized. There is an intimate connection between these sensory processes and the motor process. In those unaccustomed to reading, the motor processes are energized in the act, and even the lips may be observed to move, mental images being no doubt aroused more readily by the double than by the single process. In the act of writing, the motor processes for articulate speech are first energized, and these excite the processes for the movement of the hand, and the formation of the written symbols. Even in writing, however, it is probable that the auditory nerve-processes are revived before or with the motor processes. If you attend to the mistakes you make in writing —writing wrong letters, for instance—you may often trace the influence of these auditory and motor associations by the character of the error.

We may now ask what we know as to the parts of the brain concerned in these functions. The chief facts have already come under our notice in our review of the anatomy of the brain. In each hemisphere the lower part of the ascending frontal convolution contains the centres for the movements of the muscles concerned in articulation. From these centres, conducting fibres pass down to the lower mechanism. Hence motor processes for words must leave the cortex at this part. The adjacent posterior part of the third frontal convolution also contains structures that subserve speech, perhaps somewhat higher processes than those of the motor centres in the ascending frontal, and this region is usually regarded as the chief speech-centre. Whether the island of Reil contains similar structures is still uncertain. The first temporo-sphenoidal convolution contains the structures that subserve the auditory perception of words. Those for the visual perception of words are probably contained in or near the angular gyrus. But there is an important difference in the functions of the two hemispheres. Voluntary speech-processes go on chiefly in the left hemisphere in right-handed persons, in the right hemisphere in left-handed persons. The sensory word-processes, perhaps influenced by the motor, also go on chiefly in the left hemisphere. Disease of the left motor speech-region causes loss of the power of uttering words voluntarily; and that of the first temporal convolution, loss of the power of understanding spoken words—"word-deafness;" whereas disease of the corresponding regions of the right hemisphere produces no such effect. The power of understanding words that are seen is also localized in the left hemisphere. Thus, although the sensory centres for hearing and sight are double, one in each hemisphere, it is only in the left that they subserve the recognition of words.

And yet the left hemisphere has by no means a monopoly of speech-function. The right hemisphere contains structures of similar position and similar connections. These structures can supplement those in the left hemisphere. Loss of speech, due to permanent destruction of the speech-region in the left hemisphere, has been recovered from; and that this recovery was due to the supplemental action of the corresponding region of the right hemisphere, is proved by the fact that, in some of these cases, speech has been again lost when a fresh lesion occurred in this part of the right hemisphere. This supplemental action occurs in the sensory as well as the motor functions. It occurs far more readily in children than in adults. Permanent aphasia in children from disease of the left hemisphere is almost unknown. The loss of speech rarely lasts longer than a week. Then the child speaks almost as well as ever. Hence it is probable that speech-processes go on more equally in the two hemispheres in childhood than they do in adult life. It is also highly probable that there are individual differences in this respect among adults. Certainly, with a lasting lesion, speech is recovered more readily by some than by others. But in all persons the right hemisphere takes some share in speech-processes. Much emotional expression and automatic use of words is effected by it. This is shown by the fact that such emotional and automatic use of words remains, although the voluntary use of words is lost by disease of the left hemisphere. But since emotional and automatic expression is not lost from disease of the right hemisphere, it follows, as Hughlings-Jackson has insisted, that such expression must be effected by both hemispheres. Hence we may say that expression is one-sided, that is left-sided, in proportion as it is voluntary;

is both-sided, that is either sided, in proportion as it is involuntary and automatic. We have seen that the same law holds good of motion generally. Dr. Jackson believes that the preliminary energizing of motor word-processes that precedes utterance takes place in the right hemisphere. Perhaps, however, it occurs in both.

A curious illustration of the analogous motor impairment was presented by a man who had right hemiplegia and aphasia. In these cases there is usually more loss of simple voluntary movement of the tongue than in a similar disease of the right hemisphere, as if the speech pre-eminence of the left hemisphere carried with it a greater representation of the simpler movements of the tongue. I told the man to put out his tongue. He made many attempts, but could not. Then he put out his tongue and licked his lips—an automatic action to facilitate the process, —and tried again to put it out voluntary, but failed. An analogous speech-defect was that of a girl who, after many vain attempts to utter the word "no," said, "I can't say 'no,' sir."

The extent to which automatic word-processes may be subserved by the right hemisphere is strikingly exemplified by a case that was under my observation many years ago. A man had embolism of the left middle cerebral artery, and, as he afterwards found, the whole of the motor speech-region of the left hemisphere was destroyed. From the attack till his death, a few week afterwards, he only said "yes," "no," and uttered "ning," when the house physician wished him good-morning. But one day another patient in the ward began to sing a song,—"I dreamt that I dwelt in marble halls." The speechless patient joined in, sang the first verse with the other patient, and then sang the second verse by himself, uttering correctly every word. Of course this was not speech. No one intends to express the propositions contained in the words of the song. The words are used automatically, and this automatic utterance must have been effected by the right hemisphere.

In all cerebral affections of speech there are two elements to be distinguished: some speech is lost, and some speech is preserved, but is deranged. The loss is the effect of the destruction by the lesion; the derangement of the remaining speech is due to the imperfect action of the speech-structures that remain. The word "aphasia" is current as a general designation for all forms and degrees of loss of speech, as "anæmia" is for all forms of defect of blood. Other general terms have been proposed, equally inexact, and less convenient. Indeed, the whole subject has afforded abundant scope for word-makers, who have flooded its literature with a new terminology, to a large extent needless, and to some extent injurious, fostering a harmful tendency to divide where it is desirable only to distinguish. One recent writer alone proposes fifty special designations for varieties of aphasia.

The two most important symptoms of speech-defect correspond to the motor and sensory functions already described. (1) In some cases there is loss of the motor processes for speech. The patient can understand what is said to him, but he cannot speak, or can only use voluntarily one or two words; he cannot even repeat words. (2) In other cases the patient is unable, sometimes absolutely, to understand what is said to him. In this case, he has usually considerable power of speech, but makes mistakes in words and in their form, especially in unfamiliar expressions. The error may be so great that his speech is unintelligible. In the first case the patient is conscious of his errors; in the second he is not, because he does not understand his own utterance. These two leading varieties of aphasia are best styled (with Wernicke) motor and sensory aphasia.

But another defect is very common. There is an inability to revive voluntarily the word-images, or these are revived wrongly. The patient cannot "recollect" the word, or he recollects it wrongly. Defect of memory being termed "amnesia," this special form is termed "verbal amnesia," or sometimes "amnesic aphasia." We have seen that in the revival of words both the motor and the sensory memories take part, but the sensory (auditory) is the more important, and takes the lead. Doubtless, in educated persons, the visual memory assists; but we may, for the present, leave this out of consideration. Word-deafness involves loss of the auditory word-processes—loss, that is, of the leading mechanism for the subjective revival of words. It always, therefore, involves verbal amnesia. In motor aphasia there is much less verbal amnesia, because the sensory processes are intact; the loss of the motor memory probably causes some impairment of the power of recalling words, some verbal amnesia, although the loss is difficult to ascertain on account of the loss of speech. But verbal amnesia may exist without word-deafness, without any impairment of the power of expressing words. The patient cannot "think of a word;" but if it is told him, he utters it correctly at once. As a clinical variety of speech-defect, this form is often met with, but it is not a separate pathological form, as it has sometimes been regarded. It has been described as the loss of a special memory for words; but there is no function or seat of memory for words, except that of the word-processes, motor and sensory. Moreover, the defect in word-revival always occurs in a certain order, from the special to the general. Its indication is that the speech-processes go on in structures that are relatively inadequate. The loss is never absolute; and it is always a defect in the *voluntary* revival. Automatic utterance is largely in excess of the voluntary utterance. Conclusive evidence that it is not a distinct pathological variety is afforded by the fact that the most perfect example of this amnesic aphasia is presented by cases in which there has been, at first, complete motor aphasia, and, the lesion persisting, the patient has slowly recovered a considerable power of speech, by a re-education of the right hemisphere. Here it is manifestly a residual state, the result of the relative inadequacy of the structures in which the speech-processes go on.

We may now consider the varieties of aphasia, and the various symptoms in detail, premising that they are often combined, and the resulting symptoms are often extremely complex.

In extensive disease of the motor speech-regions, the derangement usually extends beyond the limits of vocal speech. The power of writing is lost, even when there is no paralysis of the hand. This shows, as I have already said, that in writing, the word-processes, although they must leave the cortex at the hand-centre, are first energized in the speech-centre. In severe cases, expression by gesture is at first impaired, so far as the expression of propositions is concerned. The patient nods for "no" as well as for "yes." But all power of expression is never lost. That which remains varies much in different cases, but is always the more automatic—the least special forms of expression. Emotion may be expressed by gesture perfectly, and even by words, interjections, or phrases, such as oaths, that have a propositional form but not a propositional meaning. The oath uttered in anger cannot be repeated without the emotion. Further, most patients speedily regain one or two of the lowest propositional utterances, as "yes" and "no." The word "yes" seems to be a less automatic utterance than the word "no." Children, as a rule, say "no" long before they say "yes." Perhaps these words are first regained because they are often used as interjections; they may be first regained as interjections, and this may facilitate their propositional re-acquirement.[1] These patients may also sing, as did the man whose case I have mentioned. In extensive and persistent disease of the left hemisphere, all the utterances must be effected through the right hemisphere. In rare cases, under strong emotion, a proposition may escape that is much higher than the habitual utterance of the patient, forced out, as it were, by the emotion (Hughlings-Jackson). Slowly, more power of speech returns, by the re-education of the right hemisphere; and, as I have said, more readily in some persons than in others, and most readily in children.

Motor loss, such as this, usually results from disease of the cortex; but it may also be caused by disease just below the cortex. If the disease involves the motor path some distance below the cortex, it may cause transient defect of speech; but this is soon recovered from, probably because the left region is able to act through the right by means of the commissural fibres of the corpus callosum. When the disease is just

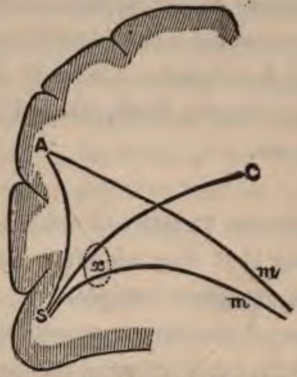

FIG. 18.—DIAGRAM OF PROBABLE COURSE OF FIBRES FROM MOTOR SPEECH-CENTRE.

A, hand-centre in the middle of the ascending frontal convolution; A *m*, fibres from this to internal capsule; S, motor speech-centre; S C, fibres from this to the corpus callosum; and S *m* to the internal capsule. S A, fibres from speech-centre to hand-centre. A lesion at *m m* will cause only transient aphasia, the speech-processes being able to pass by S C to the corpus callosum and opposite speech-region; a small lesion at *x* would cause permanent aphasia, since it involves both the fibres to the corpus callosum and internal capsule, but would not abolish expression by writing, the fibres S A (connecting the speech- and hand-centres) escaping.

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[1] "The words 'yes' and 'no' are propositions, but only when used for assent and dissent; they are used by healthy people interjectionally as well as propositionally" (Hughlings-Jackson). A child under my observation, in learning to speak, first uttered "no," and then expressed assent by the common
affirmative "m'm." This he next elaborated into "am" and for
after he had become able to speak well, he still used "am" to
When he began to say "yes," he employed it for a long time or
tion, *e. g.*, when he was called, retaining "am" for assent.

beneath the cortex these fibres are also damaged, and the aphasia is as lasting as when the cortex itself is destroyed. In the extremely rare cases in which a patient can write and cannot speak, the disease is probably so placed as to interrupt the fibres that go to the motor tract, and those to the corpus callosum, but has not destroyed the speech-centre itself, or the connection between it and the hand-centre (see Fig. 18).

The next important point to be considered is the error that occurs in the speech that still remains, or has been regained. This speech is frequently wrong, both as regards the words employed and their form. The error in form is not in articulation, but in the elements of the word. One patient, for instance, said "int" for "ink," "tinors" for "scissors"; another asked for some "pagne-cham" when she wanted some champagne. The error in form may be so great that the speech is unintelligible jargon; one patient could only say "drumlandee." The resulting disorder is often termed "ataxy of speech." Hence, this form of speech-defect is often termed "ataxic" or "atactic aphasia," an objectionable designation for the pathological variety, because equal "ataxy" may result from the opposite defect, a sensory loss, word-deafness. Moreover, the term is apt to mislead, suggesting that there is a centre or faculty for the co-ordination of speech other than the motor speech-centre.

Without error in form there may be error in use: if "yes" and "no" are the only words that can be uttered they may be used wrongly. When more speech is retained, an unintended word may be used. Errors occur, just as loss occurs, in proportion as speech is special and voluntary. There is always less error in automatic utterance and in signs. A patient who can only say "no," and utters the word on all occasions, may indicate correctly by signs, by nodding or shaking his head, whether he means "yes" or "no." It is often difficult, sometimes impossible, to understand the mechanism of these wrong utterances, but they may be, to some extent, understood by certain general principles. The first is the tendency to repetition. This depends on the fact, that a nervous arrangement, after functional activity, remains for a time in a condition in which it is more readily energized than other nervous arrangements. This often leads to error in health. If Smith has been talking to Jones
ery likely to call Jones "Mr. Robinson." It is by
that recurring utterances of what Dr. Jackson
ne established. In the speech-structures

that remain, if one arrangement has been energized, it is apt to be re-excited on every attempt to speak, and the more often the words have been uttered, the greater is their tendency to come out. It is easily conceivable that the right hemisphere may suffice for recurring utterances of low propositional value, such as "no, no." But in some patients the recurring utterance is of high propositional form, although used without meaning. The character of this utterance is often so special as to suggest, irresistibly, that it has its origin in some former utterance of the patient. Dr. Hughlings-Jackson has suggested that it is the expression that the patient was about to utter when taken ill, and that such recurring utterances occur only in the cases in which the onset was deliberate, believing that it is the result of a preliminary speech-process in the right hemisphere. I am inclined to think that it is usually the last words actually uttered by the patient, just before the attack. These cases support the view that in ordinary speech the right hemisphere takes a part in the preliminary energizing of the speech-processes, since such a recurrent utterance can only be effected through the right hemisphere. In the absolute arrest of voluntary speech-processes that follows an extensive lesion, causing aphasia, the process last energized in the right hemisphere, if energized not long before the attack, is re-energized when any attempt to speak is made and is uttered, no fresh arrangement being possible to the will for a long time. Many facts support this explanation of these recurring utterances. Thus, the recurring utterance of a signalman who was seized when on duty, was "Come on to me"; that of a girl attacked when riding on a donkey, "Gee, gee" (Hughlings-Jackson); that of a librarian was "List complete" (Russell). One of my patients was taken ill in a cab; on entering the cab she told the cabman to drive her to "Mrs. Waters," and these were the last words she spoke. Her recurring utterance was "Missis." The recurring utterance may be an unmeaning combination of syllables. Dr. Jackson has suggested that the disorder is due to the struggle of various word-processes that arise simultaneously, and of which only fragments succeed in achieving expressional predominance. Some of the error in form may also depend on the fact that relics of previous word-processes get mixed up with those that the patient intends to utter, in consequence of the readiness with which the former are re-excited. This is often obvious on an attempt to repeat the alphabet, which one patient uttered thus: *a*—a, *b*—be, *c*—ce, *d*—de, *e*—de, *f*—def, *g*—de, *h*—a, *j*—da, *k*—ka, *l*—kel, *m*—em, *n*—den, *o*—do, *p*—be, and so on. In the same way, the elements of the recurring utterances are apt to crop up when the patient begins to say other words, and so also, perhaps, are parts of words that have been familiar in previous life. Another curious and common form of error is the reversal of the syllables of a word, as in the instance of "pagne-cham," already mentioned. As I have said, words are revived subjectively before they are uttered, and it would seem that the part of the word last energized is the most vivid and most readily uttered. I have heard a very young child say "I got for," instead of "I forgot." The reversal is sometimes partial; an obtrusive consonant in the middle of the word may be uttered also at its commencement: one patient said "Lalice" for "Alice." Often the recurring utterance is a repetition of the same consonant with varying vowel signs. One patient, whenever he tried to speak, always uttered, "da, de, da, do, de, da," with extreme rapidity, like an engine-wheel flying round on a slippery rail. This is evidently explicable on the principle of repetition. What consonant is the basis of this utterance is doubtless determined by circumstances similar to those that determine the form of the recurring utterance. The woman whose chief utterance was "Missis," when she acquired some additional power of speech, and tried to utter other words, was apt to run off into "so, so, sa, se, so, si."

The loss of writing may be absolute; the patient can only make unmeaning strokes; or letters may be formed, but combined at random. Lastly, words may be written, but erroneously. The errors may be similar to those that occur in speech; repetition of words and syllables, and the substitution of one letter for another, often of one that is similar in sound, as *p* for *b*, sometimes one that is written in a similar manner, as *g* for *a*. Sometimes the order of the letters of a word are reversed; one patient wrote "tae" for "tea." The mechanism of these errors is doubtless the same as that of the similar errors that occur in speech. The same law of order of loss is traceable. It is common for a patient to be able to sign his own name when he can write nothing else, just as he can tell you his own name when he can say nothing else. A man's own signature is the most automatic of all his writing. One patient, being told to write his own name, wrote "James Slim," correctly. He was then told to write how he came to the hospital, and he wrote "cgng kgig kiyan." The errors in writing are usually greater than in speech, as indeed they are in health. In the double symbolism there is more room for error, and there is more time for error. Thus, when the patient who wrote as above was asked to *say* how he came to the hospital, he said "rail"; asked "how else?" he said "clab"; asked again, "no, way."

Patients who cannot write a word can often copy writing or copy print in printing characters, sometimes in writing characters; but they do not understand it, and copy mistakes without correcting them. I remember a sad instance of this. The son of a distinguished poetess would copy page after page of his mother's poems, although he clearly could not understand them. He would also copy, with perfect exactness, page after page of a complicated almanac—always in printing characters.

In the defect in the subjective revival of word-processes that is termed verbal amnesia, the order of loss is also from the special to the general. Proper names are lost first, and nouns are lost before verbs and adjectives. This loss for nouns has been described as a special variety of aphasia, but it is only a special example of a general and almost universal law. Among nouns, *cæteris paribus*, the more special are lost first, the more general being retained and more easily recalled. Thus, one patient, being shown a shilling, and asked what it was, replied "money," then, after a pause, "coin," and at last, after another pause, "shilling." There is often error in utterance in this form of speech-defect. The patient uses a wrong word; asks for a spoon when he wants a fork. Sometimes, with a little care, we can trace the mechanism of these wrong words. Thus, one patient with some word-deafness, but no motor loss, on being shown a pen-knife and asked what it was, said "Cornwall." Further investigation made it clear that the patient had only used a similar knife for the purpose of cutting his corns: hence the sight of a knife led to the energizing of the nerve-processes for the word "corn." He had once lived in Cornwall, and so the processes for the word "corn" led to the energizing of "wall" in association with "corn," rather than of "knife." In considerable degree, especially when there is loss of the auditory word-processes (word-deafness), there is considerable error in the forms of words, because the patient is thrown upon his motor memory, and this is apt to mislead him when it has not the co-operation of the sensory memory. No doubt this mechanism was at work in the energizing of "Cornwall," just mentioned. These patients have often an extensive power of expression by familiar phrases, if they bring them out quickly and almost automatically; if they hesitate they get wrong. "If I want to say a thing I must say it in a hurry, or I cannot say it at all," one patient said. Errors in writing are more marked in these cases than are the errors in speech, for the reason I have already mentioned, and we can often trace their origin. A patient who made no mistakes in speech wrote "disacreeable" for disagreeable, "glag" for glad. The former was evidently due to an error in the motor memory, substituting for one letter another for which the motor process is nearly the same; the second was due to the principle of repetition. The nervous arrangements are not under perfect control, and some unintended arrangement is energized, in which, for one of the reasons already mentioned, the resistance is lower. I may quote an instance of the greater readiness of familiar automatic utterance than that which is less familiar, and therefore requires a greater voluntary effort. I asked the patient who had made the remark just quoted about saying things in a hurry, to write down the name of a boat-race that had taken place the day before. He said, "Boat-race? Oxford and—Oxford and Cate—Ama—Abramidge—Oxford and Baxford—I ought to know, I have been there often—what is it?—Oxford and—now I cannot tell you—Oxford and Batham." He then took the pen and wrote "Oxford," and said, "Now I cannot say that other," and wrote "bab a tha," and then "C a t b h," and exclaimed "I am a bigger fool than ever. Oxford and Ca—Cab—Caba—Cambridge—there, I have it at last." But after repeating it correctly several times, he wrote "Cabrage."

In partial word-deafness, the same order of loss is observed. The patient may understand familiar words, and not those that are unfamiliar. He may understand signs when he does not understand speech. If told to put out his tongue, he may make no attempt to do so; but if shown he may put it out at once. Putting out the tongue is not, however, a good test. When a patient comes to a doctor, he expects to be told to put out his tongue, and may guess that this has been said to him. He may put out his tongue to order, but if told afterwards to shut his eyes, he may simply again put out his tongue. It is, perhaps, hardly accurate to speak of this as guessing. Expectation facilitates comprehension. This statement is a platitude: but the physiological aspect of the platitude is that the physical side of "expectation" is a lowering of resistance in certain nerve-processes. Hence it is better to ask the patient to do something unexpected, or, as Dr. Hughlings-Jackson suggests, to ask him some absurd question, the comprehension of which is readily shown by the obvious amusement it excites. The same condition is observed in those who are recovering from word-deafness. Slow recovery occurs from word-deafness, as from other forms of aphasia, the lesion persisting. Doubtless this also is by the re-education of the right hemisphere, perhaps aided by the visual word-centre. A very intelligent man, who had slowly recovered from complete word-deafness, told me that he still often had a difficulty in understanding a word spoken to him; but he would repeat it a few times, and at last he seemed to see the letters of the word, and then its meaning flashed upon him.

Patients with motor aphasia usually cannot read. We cannot infer from this that there is word-blindness, in the strict sense of the word. The co-operation of the motor speech-centre seems necessary for understanding the visual word-symbols. The best way of testing the power of reading is to write down a direction to perform some simple action. Patients often try to read, and even seem to think that they understand, when they certainly do not. This loss usually lessens in time, other word-processes that remain aiding the visual centre. But in true word-blindness the patient is absolutely unable to understand the words that he sees, and the loss is the more striking in that the motor processes for speech are often preserved.

I may briefly recapitulate the chief points to be attended to in examining a case of asphasia, and the indications they afford. Remember that some weeks must elaspe, from the time of the onset, before we can infer, from the character of the symptoms, the position of the lesion. The connection between the different centres concerned in speech is such that an acute lesion in one of them causes a temporary derangement of the others. Only when this has passed away, in the course of two or three weeks, can we infer the position of the lesion. The action of the speech-centres may also be interfered with, for a time, by disease that is near them but does not actually involve them, or the fibres from them.

It is well to begin by ascertaining whether the patient can understand what is said to him—whether there is any word-deafness. Try him first with simple orders, and then with sentences that are more complex. If he cannot understand even simple things, there must be disease of the first temporal convolution, or beneath it. If there is mere impairment, and not loss of auditory perception, it may be the result of partial damage to the same parts, or may be the result of old disease, from which the patient is recovering by the action of the right hemisphere.

Next observe the character of the motor loss, the degree of speech that remains, and whether the patient can repeat what is said to him. If speech is limited to a few automatic utterances, and the patient, while understanding, cannot repeat words, there is disease of the motor speech-region—third frontal and lowest part of the ascending frontal convolutions. The disease may not be in, it may be just beneath, the cortex. If utterance is only unintelligible jargon, or if there is a recurring utterance, the indication is the same. If, however, there is word-deafness, great formal error in speech does not prove disease of the motor speech-region. If the motor loss soon passes away—in the course, for instance, of two or three weeks—although hemiplegia persists, the disease is probably some distance below the cortex, in or near the internal capsule. If recovery takes place in the course of two or three months, hemiplegia persisting, the lesion is probably in the white substance near the cortex, and has damaged, but not destroyed, the fibres to the corpus callosum and opposite speech-region. This is the more probable if the patient can write better than he can speak. It is often difficult to ascertain the patient's power of writing, because the right hand is frequently paralyzed, and the left hand is awkward, while to form words with movable letters is a much more severe test than writing.

Inability to read can only be regarded as evidence of disease of the visual word-centre when there is no motor aphasia. The power of voluntarily recalling words is best tested by asking the patient to name objects that are shown to him. Defect of this may be due to past or present word-deafness, or it may be a residual condition left by a past motor aphasia. In neither of these conditions has the symptom any diagnostic value. It only indicates the relative inadequacy for voluntary action of the structures in which the speech-processes go on—in most instances those of the right hemisphere acting with those of the left that remain. If the condition is a primary one, its significance is uncertain. It is probably due to a small lesion near the sensory speech-regions of the left hemisphere, lowering their function generally, sometimes to one in the parietal lobe. There is no evidence to support the statement that is often made, that the symptom is due to, and indicates, an interruption of the connection between the sensory and motor speech-centres. Indeed, it is not easy to conceive how such an interruption could produce the effect ascribed to it.

Motor aphasia is often combined with hemiplegia from disease of the motor centres adjacent to the motor speech-region, and especially with paralysis of the face, the centre for which is the nearest. On the other hand, in sensory aphasia, hemiplegia is often absent, and, if it occurs, is usually slight.

In conclusion, one aspect of the question of speech-defect is of considerable practical importance—its relation to the capacity for making a will. In pure motor aphasia, in which the auditory word-processes are intact, words that are heard are perfectly understood, and assent or dissent can be expressed, although only by gesture, the patient could certainly make a will. If there is considerable word-deafness, it is always so doubtful whether the meaning of what is said to him is correctly perceived, that there is probably no "testamentary capacity" unless written words are perfectly understood, and all communications are thus made. *A fortiori,* if there is both word-deafness and word-blindness, a valid will could not be made.

LECTURE XI.

SYMPTOMS (*Continued*): HEADACHE—VERTIGO—VOMITING.

GENTLEMEN,—We will consider next three symptoms that stand very much on the same level of diagnostic significance,—symptoms that are often combined, although they are very different in form and in nature—headache, giddiness, vomiting. The first of these is, of all the many symptoms that organic disease may produce, the most obtrusive, the most distressing; and at the same time the most equivocal, the most apt to mislead both the patient and the physician. Local pain is a symptom of universal incidence; wherever it occurs, it absorbs the attention of the sufferer, and to him at least, suggests irresistibly a local cause. But of all seats of pain, the head is the most common, and pain is here the most suggestive of local disease, and yet it is the place in which pain is most frequent without local disease. In consequence of some mysterious relation, pain in the head is the common result of most varied causes—of stomach disturbance, of anæmia and plethora, of every kind of morbid change in the blood, febrile conditions, acute specific diseases, kidney disease, toxæmia. The nerves that supply the walls of the skull are prone to neuralgic pain of every kind. Headache occurs also in various functional disorders of the nervous system. It readily results from mechanical congestion of the brain, however produced, and is sometimes so obtrusive that the patient notices the cause less than the effect. Thus I have known a patient to come for treatment on account of headache, and to complain of nothing else, when the headache was the result of cough, and the cough of phthisis. I think it is no exaggeration to say that for one case of headache due to organic disease of the brain, you will meet with fifty that are due to other causes.

There is not much in the character of the pain to indicate, or even to suggest, its origin. Pain that follows closely the course of a cranial nerve is probably neuralgic in nature; but it may be due to irritation of the fibres of the nerve: so that, while it does not suggest, it does not exclude, intracranial disease. Pain that is very limited in area is more often of functional than of organic origin. Thus, pain limited to one supra-orbital region, or to a spot at one temple, on which the patient can put his finger, or to a spot at the vertex, is commonly of functional origin; the last is common in hysteria, and is termed, from the frequency with which it is likened to a nail being driven in, the "clavus hystericus."

Of much greater significance, though still not pathognomonic, are the course and severity of the pain, taken together. A common feature of the pain of organic disease is its constancy. The pain varies in severity from time to time, but there are rarely periods of perfect freedom. It is usually a severe pain; often at times most intense, causing the patient to shriek from the suffering. In mere severity, however, the pain of organic disease is rivalled by some functional headaches, as by that of migraine; but these are paroyxsmal—attacks of intense pain are separated by days or weeks of freedom, comparative or perfect. The pain of organic disease persists during the night, and often prevents sleep, or rouses the sufferer. Functional headaches rarely prevent sleep, which, indeed, often ends the pain. Whenever, therefore, a patient complains of severe pain in the head, ask him if it keeps him awake at night; and if it does, you should think of organic disease as possible, and search most carefully for any other symptoms. But severity and constancy of pain, even together, only *suggest* an organic cause; they do not prove such cause. Proof of this—or even the high degree of probability that, in practical medicine, we have often to be content with—is only supplied by the association of headache with other symptoms, diffuse or focal. Of the diffuse symptoms, optic neuritis and vomiting are the most significant. An ophthalmoscopic examination should never be neglected in any case of severe headache. If you can exclude three constitutional states—considerable anæmia, kidney disease, and lead-poisoning,—the coincidence of considerable neuritis and headache may be regarded as proof of organic intracranial disease. To this, and to the significance of vomiting, we shall presently return.

Always remember, gentlemen, that you can never assume the converse of diagnostic rules. The presence of a symptom may be strong positive evidence, while its absence has very little significance, and perhaps none at all. This is true in every department of diagnosis. There is hardly a disease, usually attended by some prominent and characteristic symptom, that does not occasionally occur without that symptom. Acute peritonitis, of which intense agony is a characteristic, may run its course without any pain. Neither the absence of pain in the head, nor its trifling character, enables you to exclude either intracranial disease in general, or any special form of such disease in particular. Cancer is usually a most painful malady, wherever it occurs; all tumors of the brain usually cause intense suffering: and yet cancer of the brain is occasionally almost painless. I do not say that the absence of symptoms has no negative value; it justifies you in attaching less weight to equivocal symptoms that are present, but it does not lessen the significance of any unequivocal symptoms.

We know little of the way in which pain in the head is produced. The brain of an animal can be cut or torn without the creature showing signs of suffering. But the significance of this fact may be over-rated, since other structures, that seem normally insensitive, become acutely painful when diseased. It is certain, however, that the membranes are very sensitive, and highly probable that much of the pain of intracranial disease is produced in them. Some diseases, as tumor, have been thought to cause pain by the pressure they produce, but the evidence that this is commonly the effective mechanism is insufficient, since other diseases may increase the intracranial pressure, locally and generally, without causing pain. Nor can we reasonably invoke the ready explanation of "congestion" as a universal mechanism; in many conditions of headache this is out of the question. In short, until we know more than we do at present, speculations as to the way in which headache is produced are scarcely profitable.

The question may perhaps have occurred to you—Is there any relation between the seat of pain and the position of the lesion? All that can be said is, that there is sometimes no correspondence, sometimes there is a general correspondence, and occasionally there is a close correspondence. Pain at one part of the skull is rarely caused by disease at an opposite part of the brain; one-sided pain is usually due to disease on that side; disease beneath the tentorium generally causes pain at the occiput, which may pass down the neck; and disease at the surface of the brain often causes pain corresponding closely to the seat of the lesion. Light percussion sometimes elicits tenderness over the seat of the disease—pain being produced by a tap there, and not elsewhere. This is most frequently the case when the disease is superficial. The relation of pain to the nature of the lesion we shall consider in a subsequent lecture.

Unpleasant cephalic sensations, other than actual pain, are very rarely caused by organic disease. When they occur without pain, they are almost always of functional origin, and suggest the absence of organic disease. The sensations are, however, frequent elements in the diagnostic problem, because they distress the patient, are apt to be brought on by brain-work, and, if allowed to do so, may practically incapacitate their subject. They generally occur in hypochondriacal persons, who attend much to their sensations, and to whom these feelings are fertile in suggestions of evil. Indeed, hypochondriasis often results from, when it does not cause, these sensations, and it is always increased by them. They are common in those whose brains are overworked--in clerks, in lawyers, in commercial men, and are predisposed to by neurotic inheritance, and by the condition of defective tone of the nervous system that it is fashionable now to dignify with the name of "neurasthenia," at once more alarming, and more gratifying, to the weakened mind, than the older equivalent, "nervous weakness." These sensations are various in character, but feelings of fulness in the head, heat, "rushing" sensations, and pressure on the vertex are the most common. The vertical pressure is especially frequent and troublesome. These feelings apparently depend on an abnormal consciousness of processes that should not rise into the sphere of consciousness. An infinite number of impressions must be continually reaching the brain from various parts of the body, engendered by processes of function and nutrition. Of most of these we are normally unaware, but of some we may become conscious by attention. Fix your attention on any part of the body, and in a few moments you will be conscious of some feeling there of which you were previously unaware. Let it be the vertex: in a few moments many of you will be conscious of a distinct sense of pressure there, most readily if you are tired—if you have been listening, for instance, to two or three lectures—or if you are somewhat out of health. You can understand how such a sensation, by continued attention—aided by that potent intensifier, concern—may be cultivated; the sphere of consciousness, as it were, being extended in that direction, until the sensation becomes a real trouble, and the sufferer is convinced that there must be an organic cause for that which is so distressing.

This conviction, I regret to say, is sometimes strengthened by members of our profession who find it profitable to pander to these morbid tendencies by a diagnosis of some actual disease in harmony with the patient's fancy—a diagnosis for which there is not the slightest justification. "Congestion of the base of the brain" is one of the most common of these diagnoses. There is a current manual of disease of the nervous system, in which the symptomatology of congestion of the brain has been largely written from the history of these sufferers from cephalic sensations. Endeavor to convince the patient that there is no actual mischief in brain or in circulation; encourage him to disregard the sensations, and they will slowly cease to trouble him.

There is only one condition in which these sensations, other than pain, are suggestive of real disease, and that is when they are distinctly paroxysmal and the paroxysms are accompanied by vomiting. Not long ago I saw a man who complained of occasional sensations of a "rushing" feeling passing from the occiput to the vertex. These gradually increased in intensity, until the sensation became actually painful, and with this he would sometimes vomit. There were no symptoms of organic disease, with one important exception—there was slight optic neuritis. This gave additional significance to the vomiting, and made some form of tumor highly probable. A few weeks afterwards he complained suddenly of acute pain in the head; while describing it to his doctor, he lost speech; he became unconscious, and died in a few hours. Most likely he had an intracranial aneurism, the rupture of which caused his death.

We pass next to a symptom that has excited much interest, on account of its peculiar character, its significant associations, and its relation to the disturbances that can be produced experimentally in animals. "Vertigo," I need not remind you, means "a turning." The word is strictly applied only to those sensations in which the patient feels as if he were turning round; or at least moving, or has the related objective sensation that other things are moving before him in a definite direction. But it is often, and its English equivalent "giddiness" is still more frequently, applied to all sorts of sensations that involve a sense of uncertain equilibrium, and even to vague feelings of mental confusion, or to dimness of sight—sensations that are also termed "dizziness."

Some mental confusion is no doubt always involved in the derangement of the perception of the exact relation of an individual to his environment that constitutes true "vertigo." It is not surprising, therefore, that the same term should be applied more widely to a similar mental state, irrespective of the precise mechanism by which it is produced, especially since our vocabulary is inadequate to designate, or our language to describe, a hundredth part of the varied sensations to which our frame is subject.

We are now concerned only with the sensations that do involve so distinct a sense of disturbed equilibrium or actual movement as to come under the general designation. In definite vertigo we may distinguish three elements, which are unequally prominent in different cases. The most pronounced, and most constant, is a sense of movement in the patient himself—to one side, backwards or forwards, up or down, or a sense of oscillation. Next in frequency is an apparent movement of objects at which he is looking; these may seem to move in the same direction as that in which the patient seems to move, or in the opposite direction. The third is less common than the others—an actual movement in the patient; it is usually a fall, or a visible tendency to fall; the fall is commonly in the same direction as the subjective sense of movement, but sometimes it is in the opposite direction. It is of great importance to ascertain, as exactly as possible, the precise character of each of these elements. Although we cannot, as yet, use all these facts in practical diagnosis, they enable us to understand something of the nature of the symptom, and will probably, in the future, teach us much of its mode of production.

The first significant fact is that the patient usually falls, if he does fall, in the direction in which he feels a tendency to fall. At first sight this may seem natural, even necessary; but the fact makes it probable that the sensation of vertigo is really (as Dr. Hughlings-Jackson has suggested) a motor sensation, the effect on the sensorium of motor processes, which, in greater intensity, cause an actual movement.

I told you that objects sometimes seem to move in the same direction as the patient, sometimes in the opposite direction. This difference is not very easy to explain. It has been thought that stationary objects should appear to move in the opposite direction to the subjective sense of movement, because, if the eyes move, say to one side, objects will pass across the field of vision towards the other side. Thus, if you move your eyes to the right, an object that was before in the middle of the field will pass to its left side. But our sense of the movement of visible objects does not depend only on their position, or change of position, in the field of vision; it depends on this, combined with the consciousness of our own condition of rest or movement. If, for instance, we remain still, and an object remains in the same part of the field, we conclude that it is stationary, by a process of unconscious inference, if the term may be allowed. But if an object remains in the same part of the field while our eyes are being moved to the right, we know that it is moving to the right. It is the same if the head or body is turned instead of the eyes. Hence, if we have, in vertigo, a sense of movement without actual movement, an object that remains in one part of the field seems to move in the direction of the sense of movement. When an actual movement occurs, this is probably always far slighter than the sense of movement; and the reverse movement of objects in the field, due to the actual movement of the eyes, may be insufficient to compensate for the greater sense of movement; so that they still appear to move in the same direction, although, if the patient could analyze his feelings in what is usually a moment of supreme confusion, the objects would probably seem to move less rapidly than himself. We cannot at present give an explanation of the apparent movement of objects in the opposite direction to the sense of movement of the patient. It is probable, however, that the motor and sensory relations, disturbance of which causes vertigo, are extremely complex and numerous, extending through almost all grades of motor and sensory cerebral processes, from low to high, and may be deranged at various functional levels. It may be that in certain derangements the sense of movement, even if it is attended by actual movement, does not involve visual impressions in the resulting error of judgment. Again, the tendency to movement may cause a compensatory and opposite action of the centres to preserve equilibrium, and this may, in some cases, exceed its cause. But I will not take you farther into the obscurity in which this part of the subject is involved.

The centrifugal impulses that maintain equilibrium are regulated by certain centripetal impulses that give guiding information regarding the relation of the body to its environment. One class of these consists of the various sensory impressions from the parts on which the body is resting, and of the impressions from the muscles that maintain the body in its posture (or the sense of the innervation bestowed on these muscles).

A second is derived from the innervation of the ocular muscles, and from those of the head and neck. From this, as we have before seen (p. 74), we derive, unconsciously, our perception of the relation of our body to seen objects. A third consists of the impressions derived from the semicircular canals—impressions that vary according to the pressure of the endolymph in the ampullæ of the canals, under the influence of gravitation and movement. If any one of these impressions is suddenly deranged, vertigo results. The most effective is the derangement of that derived from the ocular and aural sources of information; and the resulting derangement constitutes, respectively, ocular and aural (or labyrinthine) vertigo. The same symptom may be also produced by a very different mechanism, disturbance of the stomach.

It would be foreign to my present task to describe to you in detail all these varieties of vertigo. I mention them to you, because the symptom results from these causes far more frequently than it does from central disease, and you cannot ascribe it to the latter until you have excluded a peripheral origin. Evidence of such origin is afforded by other symptoms of derangement of the organs concerned in its production, and for these you must search. In ocular vertigo there is diplopia; in aural vertigo there is commonly the "nervous deafness" of which I described the symptoms in a previous lecture (p. 82), and there is often also a noise in the ear. In gastric vertigo there are other symptoms of stomach disturbance. Giddiness occurs also, but usually in vague form in anæmia, and other states of nervous weakness. It is sometimes met with when no cause for it can be discovered—a form which, to conceal our ignorance of its nature, has been termed "essential vertigo." Definite vertigo, in which the sense of movement is well marked, is far more frequently due to disease of the semicircular canals than to any other cause; and when it does result from any other cause, as, for instance, from stomach disturbance, you will often find some deafness or other indication of an affection of the ear,—as if the impairment of the auditory nerve had given a more definite character to vertigo that was *excited* by another mechanism. A gentleman, a member of our own profession, has suffered all his life from prostrating attacks of dyspepsia. Some years ago, he became deaf from labyrinthine disease; ever since, his attacks of dyspepsia have been accompanied by severe vertigo, from which he never suffered until he became deaf. The paroxysms of auditory vertigo may be very severe, and are often ascribed to some brain lesion. For this reason they may excite considerable, but groundless, alarm.

Another form of vertigo that may give rise to diagnostic difficulty is that of epilepsy. Vertigo is a frequent warning of the minor seizures, and sometimes of the severe attacks. The almost invariable concurrence of loss of consciousness sufficiently distinguishes this from the vertigo of peripheral origin. Remember, however, that this warning is not confined to idiopathic epilepsy. The convulsive seizures that result from organic brain-disease are often heralded by vertigo. I have already described the diagnosis between the two.

Severe vertigo, whatever its origin, is usually attended by vomiting. There must be a close connection between the pneumogastric centre and the nerves or centres that regulate equilibrium. The auditory and vagus nuclei are not far apart in the medulla, but we do not know whether the connection between the two is established there or in the cerebellum. The latter is not improbable. A connection certainly exists, and enables us to understand that stomach-disturbance may produce, and intensify, vertigo, as well as that vertigo may cause vomiting. Sea-sickness is not improbably produced by the oscillations of the endolymph in the semicircular canals.

Diseases of various parts of the brain, and lesions of various kinds, may produce vertigo, and it has therefore in itself little diagnostic significance. It is produced by chronic irritating disease, but rarely by stationary lesions. It occurs at the onset of acute lesions. The most intense vertigo, however, is caused by lesions of the middle peduncle of the cerebellum, and it may then be accompanied by its true motor form, a forced rotation.

Vomiting is occasionally a most important symptom of cerebral disease, but it is a symptom that derives its significance chiefly from its associations. It has, indeed, certain characteristics, but these, which are chiefly negative, are common to the vomiting of functional nervous disorder and of organic disease. The act of vomiting is reflex, effected through a centre in the medulla, probably part of the vagus centre. It may be produced by an undue impression on the nerve-endings in the stomach—an impression that is felt as pain, discomfort, or nausea.

Such is the vomiting of gastric origin, as that of ulcer of the stomach, or of simple indigestion. But vomiting may also occur from exalted irritability of the centre; and, in this condition, stimulation of the nerves of the stomach by the mere presence of food may be sufficient to excite the irritable centre. Such is the vomiting of hysteria, in which food is rejected as soon as it enters the stomach; and such is the vomiting of cerebral disease. The increased irritability of the centre may be so great that vomiting may occur without any peripheral irritation, when the stomach is empty.

Nausea may or may not attend this increased reflex excitability. In hysterical vomiting it is generally absent, and it is often absent in the vomiting of organic cerebral disease. We know very little about nausea. It is an effect on the sensorium of the nervous irritation that excites vomiting, but is, as it were, a collateral disturbance, and is not due simply to the action of the reflex centre, since vomiting may occur without it. It is referred as much to the fauces as to the stomach, and is prominent when vomiting is excited by tickling the fauces, and when the excitability of the centre is augmented by some other peripheral irritation, as that of the uterus. Hence, while its absence suggests, its presence is compatible with, a central cause.

Organic disease in any part of the brain will cause vomiting, and it is therefore probable that the higher central relations of the pneumogastric nerve are very extensive—a fact that is also suggested by the varied functional disturbance of the cerebral centres that may result from gastric disturbance, and by the singular readiness with which vomiting may be excited by olfactory impressions and by some psychical influences. You may perhaps know Weir Mitchell's story of the man who was so much disgusted by his wife's vomiting during her first pregnancy, that he vomited too; and ever after, when his wife became pregnant, he became sick. Although vomiting may be produced by disease in any situation, it is caused with especial readiness by disease of the pons and medulla, and most readily of all by disease of the cerebellum. It has been thought that this is due simply to pressure on the subjacent medulla, but it is probable that a functional mechanism is concerned in the effect. Of all the pathological relations of vomiting, the most remarkable is its relation to vertigo. Of this relation I have already spoken, and will only further remark that this association, coupled with the curious disturbance of equilibrium that results from disease of the cerebellum, suggests that the vomiting of cerebellar disease is due to an intimate relation between the gastric centre and this part of the brain.

Cerebral vomiting results chiefly from irritating disease. It is rarely due to a stationary lesion. Meningitis, tumor, abscess cause it; acute lesions only at their onset, or during the period of secondary irritation. There are two facts regarding cerebral vomiting that I would impress upon you. The diseases that cause vomiting usually cause also pain in the head, and the vomiting often attends paroyxsms of pain, and adds much to their significance. They give a similar significance to pain that is slight, and would not, in itself, suggest organic disease. The second fact is that brain disease, which exalts the excitability of the gastric centre, does so gradually, and the vomiting may be at first excited by a peripheral cause—a cause that might not be effective were it not for the exalted excitability of the centre. In such cases the exciting cause may be accepted too readily as an adequate explanation. This caution is especially necessary in the case of children. I have more than once known the vomiting of meningitis or of cerebral tumor to be, at first, excited by some injudicious food, and to be therefore thought to be of no importance, until graver symptoms gave significance to it. Remember that indigestion, sufficient, by itself, to make children vomit, usually makes them ill; and if a child vomits food without seeming otherwise indisposed, do not at once assume that there is nothing more the matter with it than indigestion, but watch it carefully.

LECTURE XII.

SYMPTOMS (*Continued*): TEMPERATURE—PULSE—DISTURBANCE OF RESPIRATORY, DIGESTIVE, AND URINARY ORGANS—SYMPTOMS OBSERVED WITH THE OPHTHALMOSCOPE.

GENTLEMEN,—We have to consider to-day a miscellaneous group of symptoms that are sometimes described as being "outside the nervous system," apparently because they are within the range of other organic systems. They are no more "outside the nervous system" than is paralysis of the arm. We will begin with the changes in the heat of the body that are produced by brain disease.

Elevation of temperature accompanies many diseases of the brain. The relation between the two varies in different cases. In some the pyrexia is simply associated, the result of the cause of the cerebral disease. In others it is the result of the pathological process in the brain. In others, again, it is the result of the lesion, and not of the process that causes the lesion. In each case it is necessary to recognize the precise relation, in order to estimate correctly the significance of the pyrexia. Instances of the first form are presented by the fever that accompanies embolism in a case of ulcerative endocarditis, or a secondary abscess, due to septicæmia. In these cases the pyrexia is chiefly due to the general disease, and is simply associated with the cerebral lesion. The second relation obtains in most cases of intracranial inflammation. The morbid process raises the temperature, as it would if it occupied any other situation. The pyrexia of meningitis, and the elevation of temperature that occurs a few days after an acute lesion, are instances of this form. These two causes of pyrexia often concur; a general disease causing fever produces a cerebral lesion that has a similar effect. The fever that results from an intracranial cause varies in its degree and course much more than does that which results from a similar process outside the nervous system. Two facts are especially important. Pyrexia may be absent with an inflammation that elsewhere would certainly raise the temperature. Apparently this is the effect of a restraining influence exerted by the irritated nervous system. This influence may even go so far as to lower the temperature under the normal, in spite of the process of inflammation. The second fact is that the rise in the pulse-rate which usually attends fever may, in like manner, be restrained, and this, even though the rise in the temperature is not prevented. The pulse may even be lowered to an abnormal infrequency. Thus a normal temperature does not exclude inflammation; and elevation of temperature, if it occurs, derives additional significance from an infrequent pulse.

The third relation between brain disease and temperature is very remarkable. Lesions in the pons or medulla may cause a very rapid elevation of temperature, which, in the course of an hour, may attain a height of 106°, 108°, or 110°. This hyperpyrexia is usually associated with copious perspiration. It occurs not only in various acute lesions, but sometimes in the course of chronic lesions. It may also result, although very rarely, from more extensive disease, such as meningitis, probably from an influence exerted on the lower parts of the brain. This hyperpyrexia is apparently due to the loss of action of some influence that normally restrains the heat-producing mechanisms of the body. As you may know, a similar effect follows experimental lesions in this part of the brain in animals. It is always of very grave significance, usually being followed by death within a few hours.

There are one or two miscellaneous facts regarding the temperature in diseases of the brain and nervous system with which you should be acquainted, in order to avoid errors into which you might otherwise fall. When convulsions succeed one another with great frequency—the "status epilepticus"—the temperature often rises steadily, and may attain an alarming height—106° or 108°. The rise is the result of the convulsions, and does not indicate any inflammatory condition of the brain. If the patient does not die, the temperature slowly subsides as the convulsions become less frequent. Some degenerative diseases, as general paralysis of the insane, are occasionally attended with slight chronic pyrexia, which appears to have its origin in the nervous system, and does not necessarily indicate any inflammatory condition. In the same class of diseases, paroxysmal disturbances occur, characterized by great heat of the skin, often sweating, sometimes loss of consciousness, sometimes convulsions. The patient may seem for a few hours in a most grave condition, as if some acute inflammation had suddenly developed; but in a few hours more all these symptoms have passed away. These are called "congestive attacks;" we do not know their real nature, or whether the indications of congestion are primary or secondary. Thirdly, children are sometimes liable to attacks, probably of the nature of migraine, in which there are severe headache, fever, and sometimes vomiting. The condition of the child resembles that of one suffering from meningitis, but the symptoms develop more rapidly than those of meningitis, reaching their height in a few hours from the onset. They pass away with equal rapidity. Such attacks may recur at intervals of a few weeks or months. The rapidity of the onset, and the history of previous attacks of the same character, distinguish them from more serious affections. Their recognition is important, otherwise you may make a diagnosis of fatal disease, and the patient may be well at your next visit.

Depression of temperature occurs at the onset of some acute lesions, especially hæmorrhage, and in some chronic diseases in which there is profound lethargy, and all the bodily functions are lowered, in response to the lowered function of the brain. In meningitis, the influence that prevents an elevation of the temperature may, as I have already mentioned, go so far as to reduce it below the normal, in spite of the inflammation.

Many diseases of the brain have no special influence on the pulse. The most important fact is that which I have already mentioned, that the frequency of the heart's action may not be increased, and may even be lessened, by morbid processes that elsewhere would increase the pulse-rate. In inflammation, variations in frequency occur without any recognizable condition to which they can be attributed, and irregularity in rhythm is occasionally a marked and early symptom. Infrequency is not uncommon in chronic disease, and also at the onset of sudden lesions. When the latter are in the neighborhood of the medulla, there may be great irregularity in both frequency and force.

Vaso-motor disturbance occurs as part of hemiplegic palsy, and this form has been already described. In meningitis there is often a strongs tendency to trophic disturbance in the skin—bed-sores and the like—similar to that which occurs in hemiplegia, and occasionally this symptom has a diagnostic value. I have known, for instance, the readiness with which blisters were caused by a hot-water bottle to first arouse a suspicion of the grave nature of an illness that had been regarded as hysteria. Dilatation of the cutaneous vessels, excited by an irritation of the skin (such as a stroke with the finger-nail), lasts long in many cases of acute brain disease, and much significance was formerly attached to to the "tache cérébrale," as the red line thus produced was called. But it occurs also in many general diseases, and is as likely to mislead as to aid the diagnosis.

Respiration may be either quickened or slowed by cerebral disease; but the change is considerable only when the functions of the brain are impaired by extensive disease, such as a large hæmorrhage, or when the lesion is in the neighborhood of the respiratory centre, *i.e.*, is in the pons or the medulla. Under these circumstances the respirations are sometimes very irregular, and there is occasionally a peculiar respiratory convulsion, a prolonged convulsive cough, or a hiccough. The rhythmical variations that constitute the "Cheyne-Stokes" breathing (see p. 95) occur towards the end of many cerebral diseases, such as cerebral hæmorrhage or meningitis. In some rare cases of disease of the pons or medulla, in which consciousness has been preserved, there has been intense subjective dyspnœa.

In coma, the respirations are often quickened, not as a direct result of the cerebral lesion, but in consequence of the accumulation of mucus in the air-passages. This is due chiefly to respiratory inertia, but in some cases neurotic congestion may increase the secretion, since lesions near the pneumogastric centre may cause actual hæmorrhage into the substance of the lung.

Of the derangements of the digestive organs, the most important, vomiting, has been already described. Constipation is common in acute and subacute diseases, and sometimes adds weight to the significance of other symptoms.

Alterations in the composition of the urine may be a direct result of the cerebral lesion, but are rare, and of little diagnostic value. Excessive secretion, and the presence of albumen and sugar, have been observed as the result of disease, acute and chronic, in various parts of the brain, most frequently when the lesion was in the pons. The derangement has usually been transient; occasionally, glycosuria has continued for many months. I need scarcely mention that organic renal disease, and previous diabetes, must be carefully excluded before the change in the urine can be ascribed to the influence of the cerebral lesion.

The sphincters are affected as frequently in disease of the brain as in that of the spinal cord, but rarely in the same degree. Both retention of urine and incontinence are met with, but the incontinence is the result of overflow, and not of complete inaction of the sphincter. It is very important to remember how often there is an apparent incontinence that is merely the result of mental inertia. I have mentioned this before, in speaking of the mental changes; and I told you how significant it is, as an indication of a profound degree of mental change—more profound than the other symptoms might lead you to imagine.

I have left to the last the changes in the fundus oculi, not because they are last in importance, but because they stand to the cerebral lesion in a relation somewhat different from that of the symptoms that have hitherto engaged our attention.

The indications of brain disease that are revealed by the ophthalmoscope are not only of the highest practical importance, but are of interest as a salient indication of the progress of medical science, since thirty years ago they were unknown. Morbid appearances in the eye are frequent in intracranial disease, and often prove the existence of such disease when other symptoms are inconclusive, or afford evidence of its nature that can be obtained from no other source. For this and other reasons, skill in the use of the ophthalmoscope, and familiarity with the conditions it reveals, are indispensable to the physician. I cannot too strongly impress upon you the importance of acquiring dexterity in the use of the instrument as early as possible, in order that you may be able to utilize the opportunities for gaining experience which almost every department of practical work will supply. These opportunities will be lost if you only learn to use the instrument when you proceed, towards the close of your course, to the special study of diseases of the eye.

Some of the changes in the eye met with in brain disease are merely associated, the result of, and evidence of the cause of, the cerebral lesion. Others are consecutive, the effect of the brain disease, and evidence of its presence and of its nature. The associated and consecutive symptoms are sometimes conjoined.

The associated changes are due to the constitutional condition on which the brain disease ultimately depends. The most important of these are the following: First, albuminuric retinitis, acute or chronic, associated chiefly with arterial degeneration, such as causes cerebral hæmorrhage or cerebral softening. Secondly, syphilitic disease, especially choroiditis, or choroiditic atrophy, associated with lesions of the brain due to acquired or inherited syphilis. Thirdly, tubercular disease—tubercles of the choroid occasionally in tubercular meningitis, very rarely massive tubercle in cases of tubercular growths in the brain. Other forms of associated growth are too rare to be of practical importance. Fourthly, vascular lesions of the retina sometimes coincide with similar lesions in the brain. Thus, embolism of the central artery of the retina may occur in the same patient, and even at the same time, as embolism of a cerebral artery. Miliary aneurisms on the retinal arteries have been observed to coincide with cerebral hæmorrhage due to similar aneurisms in the brain. They are, however, extremely rare. Hæmorrhages in the retina are more common, and are of some significance. They occur especially in albuminuria, in gout that has profoundly affected the system, in leucocythæmia, profound anæmia, purpura, ulcerative endocarditis, and other forms of septicæmia. In the latter they have often white spots in the centre, and are the result of capillary septic embolism. Hæmorrhages, identical in aspect, are sometimes found in the same cases in the cerebral meninges.

The consecutive changes, that are the result of the cerebral disease, comprehend optic neuritis and atrophy. Neuritis or papillitis consists in swelling and increased vascularity of the intra-ocular termination of the optic nerve—the "optic papilla." It will be remembered that the end of the nerve is visible in the fundus as the "optic disc," the boundary of which is the oval opening in the sclerotic and choroid. The fibres radiate from the nerve on all sides, but not equally; they are few on the temporal side. The nerve-elements are so translucent that they do not obscure the edge of the disc, except in some cases and to a slight extent, above and below, where they are more numerous. The disc is rarely uniform in its surface, because the separation of the nerve-fibres leaves a central hollow—the "physiological cup"—in which there are few or no vessels, and which is therefore pale, while the periphery of the disc has a rosy tint from the minute vessels that lie among the nerve-fibres. This physiological cup varies much in size, and may be absent. The vascular portion of the disc also varies in extent (inversely as the cup), and varies so much in tint that no inference can be drawn from its color unless this is observed to change from time to time. Mere congestion, therefore, consisting only of increased vascularity of the disc, is very difficult to recognize, and, although it probably occurs, it is extremely rare as an isolated condition, and is of small practical importance. An actual pathological change, however slight in degree, is usually attended by some swelling of the papilla, and especially by lessened transparency of its structures. The effect of this change is first to lessen the sharpness of the edge of the disc, and then to obscure it altogether. It is, therefore, to this point, the sharpness of the edge, that attention must chiefly be directed. The effect of a morbid change on the edge of the disc is greater when the examination is made by the direct than by the indirect method. On the other hand, if the indistinctness is apparent, and not real—is due to the tint of the disc being nearly that of the adjacent choroid, and not to the lessened transparency of the structures in front of it—the edge is more distinct by the direct than by the indirect method of examination. In the early stage of neuritis the edge of the disc, seen by the indirect method, may appear a little blurred, and surrounded by a pale halo, while by the direct method the halo is resolved into a striated, semi-opaque layer, completely concealing the edge. The early change is greatest on the nasal side, which may be obscured when the temporal edge of the disc, on which there are few nerve-fibres, is still sharp. As the change advances, it involves the whole circumference of the disc, and the swelling rapidly encroaches on, and ultimately obliterates, the normal white depression in the centre of the disc. The prominence of the swelling is readily recognized by the relative displacement that the different parts appear to undergo on a lateral movement of the observer's head in the direct, or of the lens in the indirect, method of examination. It is also shown by the loss of the central reflection from the vessels where they course down the sides of the swelling, and their plane is no longer at right angles to the line of vision. The tint of the swollen papilla becomes a full red, or, more commonly, a grayish-red, on indirect examination, but the direct method shows a fine striated vascularity. As the prominence increases, the swelling becomes wider in extent, until it may be two or three times the diameter of the normal disc. White spots may appear on its surface, due to accumulation of products of degeneration. At first the retinal vessels present little change in size, but as the swelling increases, their compression causes the veins to become broader and the arteries narrower, and extravasations of blood may be visible on the surface or margins of the swollen area. The process varies much in the rapidity with which it is developed; it may reach a considerable intensity in a fortnight, or be still moderate in degree at the end of three or four months. As a rule, the more quickly it is developed, the more intense it becomes. Retrogression is indicated by a diminution in the vascularity, still greater contraction of the arteries, and, later on, contraction of the veins also, if the degree of neuritis has been considerable. The swelling remains, for some time, pale and soft-edged, and slowly sinks until the edges of the disc appear. The substance of the disc is for long, often permanently, occupied by new tissue, which, with the narrowed arteries, affords evidence of the preceding inflammation. Other indications of this are often to be seen in damage to the adjacent edge of the choroid. It is only when the neuritis has been slight that the disc resumes a perfectly normal aspect. When much new tissue has been formed, this, by its cicatricial changes, leaves the disc white and atrophied, in the condition of "consecutive atrophy," or "neuritic (papillitic) atrophy," as it is also termed. When the papillitis has been very intense, and the swelling wide in area, the adjacent retina may suffer in its nutrition, and products of degeneration may remain as white spots, especially near the macula lutea, simulating the aspect of albuminuric retinitis. The microscope reveals, in the substance of the swollen papilla, various inflammatory changes, of which you will find details and illustrations in my work on "Medical Ophthalmoscopy." Similar, although slighter, changes may be traced back in the substance of the optic nerves, or in their sheaths, even to the chiasma, and they may be more intense in front of the chiasma than they are midway between this and the eye. The outer sheath of the optic nerve in most cases is distended by liquid, so as to form a pyriform swelling behind the globe. The space within this sheath is continuous, behind, with the subarachnoid space around the brain, and in front, with the lymphatic spaces in the optic papilla.

A slight and even considerable degree of optic neuritis may cause no symptoms; acuity of vision may be unimpaired, color-vision normal, the visual fields unrestricted; but when intense, sight suffers in each of these characters, and may be entirely lost. The damage to vision is often greater during the stage of subsidence than it is during the active period of inflammation, probably because the nerve-fibres are compressed by the cicatrical contraction of the tissue-elements formed during the inflammation. As I mentioned in a previous lecture (p. 64), the affection of sight is not necessarily due to the visible papillitis; it may be the result of more intense inflammation behind the eye, or the effect of the intracranial disease. After the neuritis has quite subsided, improvement in sight often takes place by the recovery of some of the damaged fibres, but there may remain absolute blindness, or considerable amblyopia, and often there are very irregular changes in the field of vision, both for white and colors.

Optic neuritis may result from many disease of the brain, but the most frequent cause is tumor, and a considerable degree of neuritis is seldom due to any other lesion. Next in causal frequency is meningitis; especially when it affects the base of the brain; the inflammation caused by meningitis is usually less intense than that of tumor, perhaps on account of the briefer duration of the disease. Optic neuritis does not result from cerebral hæmorrhage, or from thrombotic softening. It has been several times observed in cases of softening from embolism, when the source of the obstructing plug was active endocarditis, perhaps because the material carried from the valves is of an irritating, septic character, and determines a greater degree of secondary inflammation in the softening produced. Neuritis has also been met with, once or twice, in cases in which no naked-eye lesion of the brain could be found, but in which the microscope revealed slight inflammatory changes—a diffuse cerebritis. In all these cases the neuritis is, as a rule, double, although it may develop more rapidly in one eye than in the other. Occasionally it is one-sided, and then it is generally on the side opposite to the lesion; but unilateral optic neuritis is much more frequently due to disease at the back of the orbit, or at the optic foramen.

It must be remembered, however, that optic neuritis may result from other causes than intracranial disease; it occurs in chlorosis, albuminuria, lead-poisoning and after certain fevers, especially scarlet fever and typhoid. An important fact is that in many of these conditions its occurrence is associated with some cerebral symptoms; thus, when neuritis preponderates over the other retinal changes in albuminuria, there is usually much pain in the head, and in lead-poisoning it is usually associated with the acute cerebral symptoms that have received the name of "encephalopathia saturnina." Remember this concurrence of cerebral symptoms with neuritis due to a general disease, because it often gives rise to a diagnostic difficulty.

The mechanism by which optic neuritis is produced is a subject on which various opinions have been held. A full account of these will be found in "Medical Ophthalmoscopy."[1] It is sufficient here to say that the early theory that neuritis is due to increased intracranial pressure, acting mechanically, has been proved erroneous. Slow increase of intracranial pressure has almost no influence on the occurrence of this symptom. In a large number of cases of neuritis there is distinct evidence of a descending inflammation, either along the trunk of the optic nerve, or along its sheath; and in cases of meningitis, such descending inflammation is invariable. The distention of the optic sheath with serum has been regarded as the chief mechanism, but it is not essential for the production of neuritis; it may be absent, and its occurrence is related especially to the presence of an excess of subarachnoid fluid. The signs of mechanical "strangulation," which are to be observed in cases of intense neuritis, are no evidence that the inflammation was caused by any mechanical process. The cause of the strangulation is the compression of the veins by the inflammatory products within the swollen papilla, and not, as was once thought, their compression within the sclerotic ring or behind it by the distention of the sheath. It is probable that optic neuritis is rarely due to a single factor, and that the most potent element is the descent of a process of tissue-irritation, which, when it reaches the papilla, sets up a more intense inflammation; that in some cases this influence is alone effective; and that in others it is aided by the distention of the sheath, which hinders the escape of effete products, increases œdema, or even conveys irritating material. The distinction between optic neuritis and "choked disc" is one of degree, and not of mechanism. So far as optic neuritis has any single significance, it is that of an irritative process within the skull.

There is an important relation between the chronicity of the neuritis and that of the intracranial process. A chronic cerebral process may cause an acute neuritis, but a chronic neuritis never results from an acute process, and the degree of the chronicity of the neuritis is an indication of the degree of chronicity of the intracranial disease. When the latter begins to improve, the neuritis lesssens, and the commencing subsidence of the intra-ocular inflammation is often the first indication of the improvement of the brain lesio

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[1] Second Ed., p. 65.

Optic nerve atrophy is a less frequent symptom of brain disease than is optic neuritis, with the exception of that form of atrophy which is the consequence and evidence of past inflammation. Primary atrophy is rare, and confined to those forms of brain disease that are degenerative in nature. It is met with, for instance, in cases of disseminated sclerosis, and of general paralysis of the insane, as an associated symptom. Secondary atrophy occurs, not infrequently, in consequence of damage to the optic nerve behind the eye, or to the optic chiasma. Disease of the optic tract does not cause marked atrophy of the disc. When the disease is at the chiasma, both eyes may suffer; when it is in front of the chiasma, the atrophy is usually limited to one eye. The characteristic of secondary atrophy is that sight fails without ophthalmoscopic changes to account for the failure, and this is slowly followed by atrophy. The only distinctive features of the failure occur when the disease is at the chiasma (see p. 64). If the lesion of the nerve is inflammatory, some descending inflammation may reach the eye and be visible at the optic disc. Its amount depends on the intensity of the inflammation, and on its proximity to the eye, but it is always insufficient to account for the affection of vision. When the lesion is near the eye, the vessels often present distinct constriction. Of the significance of these various changes as indications of the pathological nature of the cerebral lesion, I shall have more to say when this problem engages our special attention.

You may have observed that, in this outline of the changes met with in brain disease, I have said nothing about congestion or anæmia of the retinal vessels or of the disc, as an indication of similar states of the brain. Exaggerated expectations and, indeed, opinions were formed, when the ophthalmoscope was first introduced, regarding its revelations. It was thought that, since the blood comes to the eye from a cerebral artery, and returns to an intracranial sinus, the circulation in the eye would share, and show, the changes in that of the brain. But experience has not confirmed the opinion. The circulation in the eye is, so to speak, autonomous; the state of the vessels is regulated by independent influences. When all the veins of the head become over-distended, in consequence of an obstruction to the flow through the superior vena cava, the retinal veins suffer far less than those on the surface of the head, no doubt because, in the closed chamber of the eye, neither distention nor contraction of the vessels can readily occur from a mechanical cause. The information afforded by the ophthalmoscope is important and extensive beyond the dreams of its inventors, but the expectation that was most confidently held has not been fulfilled; and if you find any author professing to diagnose the state of the cerebral circulation from that of the eye, or drawing therefrom indications for treatment, you may safely conclude that he knows very little about the subject.

LECTURE XIII.

DIAGNOSIS OF THE SEAT OF DISEASE: "LOCALIZATION"—GENERAL METHOD OF DIAGNOSIS—CEREBRAL HEMISPHERE, CRUS, THE CORPORA QUADRIGEMINA.

Gentlemen,—According to the loose use of words that is still common in medical terminology, we are said to "localize" disease, that is, to make it local, when we infer its locality from the symptoms it produces; and this process of inference is termed "localization." In the previous lectures we have considered the chief symptoms of brain disease, and we have learned in what part of the brain a lesion may be situated to produce those symptoms. It is necessary now to review these facts from the other side; to look at the symptoms from the standpoint of brain regions. Before we do so, I must remind you of the distinction between the direct and indirect effects of disease (see p. 41), and that only those symptoms which last for some time can be regarded as due to the destruction wrought by the morbid process. Moreover, you must bear in mind that the nature of a lesion, as well as its position, exerts an influence on the character of the symptoms that are produced. Speaking generally, we may say that in acute lesions the symptoms are, at first, wider in range, while in chronic lesions they are, throughout, slighter in degree, than might be anticipated from our knowledge of the function of the parts that are implicated. Moreover, the symptoms produced by slow processes, such as a growth or an abscess, differ in range, according as the disease influences the gray or the white substance, nerve-cells or nerve-fibres. The fibres are strangely tolerant of slow pressure, especially when this is widely diffused. A tumor or an abscess may occupy the position of the motor path, and yet cause only slight palsy, because the fibres have been merely displaced by it, and still retain much power of conducting, in spite of their compression. When such a disease occupies the gray matter, there is a similar, although slighter, tolerance of pressure, so far as concerns loss of function, but the nerve-cells exposed to the morbid influence are in a state of abnormal irritability, and the connection between the nerve-cells of the cortex is so abundant and extensive, that the functional change may spread over a wide area and to a considerable distance from the primary disease. Discharges, causing convulsions, may commmence at various parts of the area thus exalted in irritability. Moreover, centres are sometimes inhibited by the irritation, instead of being "discharged;" and there results loss of function instead of over-action. Hence, the symptoms produced by such diseases are often anomalous, and extreme care is needed in drawing conclusions from them. Some extensive collections of facts relative to this subject have been rendered absolutely valueless by disregard of this consideration.

To return to our immediate subject. Having ascertained what symptoms are present, we must, in the case of an acute lesion, endeavor to distinguish the direct from the indirect. We can only do this with certainty by waiting till the indirect symptoms have passed away, as they will have done in a few weeks. We may, indeed, make a guess at the direct symptoms soon after the onset, from their severity, and from the absence of any indication of early improvement in them; but to attain a high degree of probability in our local diagnosis we must, in many cases, wait. Fortunately we have not thus to wait for indications of the nature of the lesion, on which, and not on the exact seat of the disease, our treatment depends.

Before you can draw any conclusion from the symptoms as to the seat of the disease, you must consider their mode of development, whether slow or sudden, simultaneous or successive. The bearing of this on the problem we are now considering is very important. If the morbid process is one of sudden onset, only those symptoms that came on together can be regarded as due to the same lesion. Take, for example, a not uncommon case, that of a patient who presents paralysis of the limbs on one side, and of the third nerve on the other. These are symptoms of disease of the crus. But to have this significance, if the hemiplegia came on suddenly, the paralysis of the third nerve must have come on at the same time. If you find, on inquiry, that the paralysis of the third nerve existed before the sudden hemiplegia, it cannot be due to the same morbid process, and the affection of the third nerve must be eliminated from the problem of the seat of the disease causing the hemiplegia. But if both were gradual in development, we cannot certainly dissociate them; they may then be due to one lesion or to separate lesions.

Having thus determined what symptoms developed in such a manner that they may possibly be due to a single lesion, we have next to ask, Is there any part of the brain in which a single lesion would produce this group of symptoms? Some combinations of symptoms can be caused only by a lesion in one situation; others may be produced by a lesion in any one of several places. Others, again, are of such a character that there is no part of the brain in which a single lesion will produce them. To return to the instance we have just taken, hemiplegia on one side, and paralysis of the third nerve on the other, can be produced only by a lesion of the crus. Hemiplegia and paralysis of the third nerve on the same side cannot be caused by a single lesion in any situation, since the motor tract, where contiguous to the nerve, is that to the limbs of the opposite side.

Many of the symptoms of brain disease are of no localizing value, because they may be caused by disease in any situation; while others are of low value, because, although produced by disease in various places, they are rather more frequent when it occupies certain situations. Most of the diffuse symptoms (see p. 42) are of this character—headache, vomiting, optic neuritis, mental change, loss of consciousness. In so far as these vary with the seat of the disease, their variations have been mentioned in the account of the symptoms. Some symptoms that are focal, and not diffuse, are produced by disease in so many places that they have but little significance, as, for instance, conjugate deviation of the head and eyes from the paralyzed side in severe hemiplegia. This may occur in lesions of the motor tract anywhere between the pons and the cortex.

Bearing in mind these facts, we may proceed to our review of the chief symptoms that are produced by lesions in various parts of the brain, and we may commence with the *cerebral cortex*. In the *frontal lobe*, or rather what is termed the prefrontal lobe, in front of the ascending frontal, lesions are usually unattended by either motor or sensory symptoms. Considerable mental change is rather more frequent than in other situations, sometimes taking the form of chronic insanity, and gives some support to the opinion which ascribes to this region a high psychical importance. But these symptoms are neither characteristic nor invariable.

The posterior part of the lowest frontal convolution on the left side constitutes, however, an exception to the negative character of this region, since disease here, around the anterior limb of the fissure of Sylvius, causes, with much uniformity, motor aphasia. Lesions which irritate, and are adjacent to the ascending frontal, often cause convulsions that begin locally. It is possible that the limb centres are not always strictly limited to the ascending frontal and that disease on the roots of the antero-posterior gyri may cause motor symptoms in some individuals, and not in others; but no symptoms have been observed in man corresponding to the functional centres that you often see marked on these convolutions in diagrams of the human brain, to which they have been transferred from the brains of monkeys.

In the *central area* of the brain (as the motor region—ascending frontal, ascending parietal, and superior parietal—is often termed), disease causes motor paralysis. There is loss of power in the leg when the lesion is in the upper third; loss of power in the arm, when in the middle third; in the face, when the disease is in the upper half of the lower third; in the lips and tongue, when it is in the lowest part of the ascending frontal; and, on the left side, disease in this part causes also aphasia. Partial hemiplegia is much more common than paralysis involving the whole of the side, and the parts that suffer together are those for which the centres are contiguous in the cortex. Convulsions are exceedingly common, and their characteristic feature is the local commencement, already described, and due to the commencement of the discharge in an irritated centre. This is not always the region most diseased. Considerable disease in one centre may cause discharges to start from an adjacent centre less diseased, and therefore capable of greater functional excitement. Thus a tumor occupying the highest part of the ascending frontal convolution (leg-centre) caused at first convulsions beginning in the foot, and afterwards, as the disease advanced, convulsions beginning in the hand (the centre for which was not invaded by the growth), and even sometimes in the face (the centre for which was some distance from the tumor). Thus, also, the disease causing such convulsion may be near, but not in the motor area. Hence local paralysis is a much more decisive indication of the seat of the disease than are the local convulsions. In the case of tumor, with changing convulsions, just mentioned, the leg became paralyzed, and this indicated that it was in the leg-centre that the destruction of tissue was in progress. Disease of the motor cortex often causes some defect of sensation, chiefly in the extremity of the paralyzed limb, and in the convulsions a local sensory aura often precedes the spasm, may even pass through the whole side before the spasm comes on (see p. 54). There is never complete hemianæsthesia when the disease is limited to the motor region. With the cutaneous defect in the extremity paralyzed, and even without this, there is usually an inability to recognize the position of the limb; but we do not yet know whether this loss is confined to cortical lesions. Probably it is not (see p. 61).

Disease is rarely limited to the remaing part of the *parietal lobe,* supra-marginal convolution, and there is much uncertainty as to the symptoms produced. The lesion usually extends to the motor region, causing hemiplegia, and there is some reason to think that ptosis accompanies the hemiplegia rather more frequently when the lower parietal lobe is affected. Extensive disease of the outer surface of the hemisphere causes hemianæsthesia, and to this the disease of the lower parietal lobe probably contributes. Defects of speech have been met with, although not with such constancy or uniformity as to furnish diagnostic indications. A lesion of the angular gyrus at the infero-posterior angle of the parietal lobe, probably causes crossed amblyopia, and disease about this region causes word-blindness; but the precise locality on which this symptom depends is not yet known. Only one symptom is known to be produced by disease of the *occipital lobe*—hemiopia,—and we are still uncertain as to the exact part of the cortex of the occipital lobe on which the symptom depends. Small lesions may cause a loss of only part of the half-field, *e.g.*, a quadrantic defect. It is probable that the separation between the preserved and blind halves of the field does not differ from that in the hemiopia produced by disease in the lower part of the visual path, but that there is greater peripheral limitation of the preserved half of the field. Irritating disease of this region may cause convulsions that commence with a visual aura referred to the opposite eye or opposite side. Disease of the *temporo-sphenoidal* lobe causes no motor or sensory symptoms unless the first temporal convolution is involved, and then deafness is produced on the opposite side; and if the disease is on the left side of the brain, there is word-deafness and sensory aphasia. Irritating disease may cause convulsions that commence with an auditory aura. If the lesion is on the inner surface of the lobe, and involves the anterior part of the uncinate convolution, it is probable that loss of smell may occur on the same side. It is important to remember that the inner part of this lobe is contiguous to the optic tract; tumors are apt to invade the tract, and thus cause hemiopia, which may be followed by hemiplegia from the further extension of the disease into the crus cerebri. This is one of the commonest causes of hemiopia from disease of the tract.

The *islana of Reil* is rarely the seat of isolated lesions, and much uncertainty still exists regarding its functions and the symptoms that result from its damage. Defects of speech, similar to those that result from disease of the third frontal convolution, have been attributed to its disease; but the evidence is not altogether satisfactory, and speech-defect has been absent in some cases in which the insula was extensively damaged.

In the *white substance of the hemisphere*, centrum ovale, the symptoms produced by disease resemble, for the most part, those that are caused by lesions of the corresponding part of the cortex, with the exception that convulsions are rare, save in the cases of tumors, which cause persistent irritation. The disturbance of speech is transient unless the disease is seated just beneath the cortex, for the reason given in a previous lecture (p. 112). Hemiopia results from disease of the white substance of the occipital lobe, but it is doubtful whether crossed amblyopia occurs unless the angular gyrus is affected. Lesions of the white substance of the lower part of the temporal lobe, and of most of that of the pre-frontal lobe, cause no symptoms.

In the *central ganglia*, lesions of either part of the corpus striatum cause no lasting symptoms if limited to the gray matter. I have seen the lenticular nucleus softened from one end to the other, and the patient, before death, presented no indication of hemiplegia. I will not say that the disease caused no symptoms, but her history was very carefully taken, and no account was obtained of any motor or sensory symptoms. Such cases are, however, extremely rare, because, in most instances, the internal capsule suffers also. A lesion of the anterior or middle part of the optic thalamus may also cause no symptoms, although there is some reason to think that the latter sometimes causes athetoid inco-ordination. When the posterior portion of the thalamus, the pulvinar, is diseased, hemiopia occurs; but it is not quite certain whether it depends on such disease, or whether there is, in these cases, interference with the posterior portion of the optic tract, which enters the corpus geniculatum close to the posterior extremity of the thalamus. The balance of evidence, however, is in favor of the dependence of the symptom on the disease of the thalamus.

The effects of disease of the *internal capsule* vary according to the part damaged. When this is the anterior limb, between the caudate and lenticular nuclei, no symptoms result. Disease of the anterior two-thirds of the hinder limb causes hemiplegia of the ordinary type, and is indeed the most frequent cause of this. It is supplied by branches of the middle cerebral that are apt to be occluded, either from disease of the wall of the vessel, or from embolism, and the arteries adjacent rupture more frequently than any other, causing hæmorrhage, which tears across the fibres of the capsule. Since the fibres for the face occupy, as we have seen, the junction of the two parts of the capsule, an isolated facial palsy may result from a very small lesion in this situation; more frequently the adjacent fibres for the arm are involved also. The leg-fibres, lying behind the others, may escape in a very small lesion paralyzing face and arm, while damage to the hinder part of the posterior limb causes a preponderant paralysis of the leg. If the disease involves the hinder third of the posterior limb, occupied by the sensory path, hemianæsthesia is produced—loss of sensation on the opposite side, head, trunk, and limbs, extending up to the middle line. The loss is sometimes complete; more often a touch is unfelt, while a strong painful impression is perceived. The special senses are also often involved—taste, hearing, and perhaps smell being lost on the anæsthetic side, while vision to that side is lost in each eye (hemiopia). The last may result either from disease of the external corpus geniculatum, or from damage to the fibres from the tract to the cortex, or from damage to the optic thalamus, or from the latter to the cortex. Considerable damage to one part of the capsule is often attended by slighter and transient damage to the other part; hence, hemianæsthesia may occur at the onset of hemiplegia and pass away, and, conversely, hemianæsthesia is often accompanied by transient weakness of the limbs on the anæsthetic side. Convulsions rarely result from disease in this region. Incomplete hemiplegia is, however, often accompanied by mobile spasm (athetosis, post-hemiplegic chorea), but we do not know whether this is to be ascribed to the disease of the capsule, or to that of the adjacent ganglia, especially of the optic thalamus.

The *corpora quadrigemina* are so rarely damaged alone, that there is much uncertainty as to the symptoms that depend upon their disease. The balance of evidence is strongly against the old opinion that sight is impaired. The chief effect is probably derangement of the internal and external ocular muscles, sometimes impairment of certain ocular movements, especially of the upward movement of the eyes and elevation of the lids. An inco-ordination of movement, noted in some cases of tumor, is probably the result of pressure on the adjacent middle lobe of the cerebellum.

Damage to the *crus cerebri* causes paralysis of the face and limbs on the opposite side, similar in character to that which results from disease of the internal capsule, but accompanied by palsy of the third nerve on the side of the lesion, and therefore on that opposite the hemiplegia. Hemianæsthesia may accompany the motor palsy, from implication of the sensory path in the tegmentum of the crus. Hemiopia may result from damage to the optic tract, but is very rare, since the lesion is usually situated close to the pons. Giddiness is common, but is usually ocular, and due to the affection of the third nerve.

These facts of localization are complex, and those that remain are still more so. It may be well, therefore, to postpone their consideration until our next meeting.

LECTURE XIV.

DIAGNOSIS OF THE SEAT OF DISEASE (*Continued*): PONS, MEDULLA, CEREBELLUM; BASE; VENTRICLES—REVIEW OF SYMPTOMS IN RELATION TO LOCALITY—BASAL LESIONS.

GENTLEMEN,—In the last lecture, you will remember, we passed in review the symptoms that are produced by disease in various parts of the cerebral hemispheres, in the crus cerebri, and in the corpora quadrigemina. To-day we resume our survey, and must notice first the effect of lesions in the mesencephalon, where the paths from the hemisphere to the limbs are brought into proximity, where the chief cranial nerves have their origin, and where an important junction is effected between the fibres of the cerebellum and those of the cerebral hemisphere.

In the *pons Varolii* a lesion usually causes very characteristic symptoms, due to the implication of the motor path and of the cranial nerves that arise from the pons. Tumors that grow slowly, however, and especially infiltrating tumors, may attain a considerable size without causing characteristic symptoms. These symptoms may be either unilateral or bilateral, according as the disease affects only one or both sides of the pons. Motor palsy is very common. Hemiplegia results from one-sided disease, the limbs being, of course, paralyzed on the side opposite the lesion. In the upper half of the pons, the upward facial path having crossed, the face may suffer with the limbs exactly as in disease of the internal capsule; but the chief characteristic of pontine hemiplegia is the affection of the fifth, facial, or sixth nerves on the side of the lesion, that is, opposite to the limbs, constituting the "alternate hemiplegia" already described (see p. 50). The tongue suffers as in ordinary hemiplegia. The palsy of the fifth may involve motor or sensory parts, or both; in the latter case there is usually much pain, referred to the face, and due to irritation of the nerve-fibres. When there is crossed palsy of the face, this is usually unaffected on the side of the limb-palsy, but extensive disease may affect both sides of the face. Paralysis of the sixth may be associated with weakness of the opposite internal rectus, causing a paralytic inability to move the eyes towards the side of the lesion; they are thus in conjugate deviation towards the paralyzed limbs, a direction opposite to that usually caused by disease of the hemisphere. In bilateral disease, various combinations of paralysis of limbs and the cranial nerves may occur. In acute lesions of the upper part of the pons, the pupils are often strongly contracted from irritation of the nuclei of the third nerve, and, if these nuclei are invaded and destroyed, the contraction gives place to dilatation and immobility. Difficulty in swallowing and articulation result from interference with the adjacent medulla. Convulsions are rare, except in acute lesions, and are then sometimes peculiar, affecting both arms or both legs. Tonic spasm in the paralyzed limbs is a common and often very marked symptom. Rhythmical movements are rare. Anæsthesia is less common than motor palsy, and the two are often disproportioned. The explanation of this lies in the fact that the motor path is in the anterior part of the pons, the sensory in the outer part of the posterior portion, the two being separated by the deeper transverse fibres. Tingling and formication in the limbs are occasionally marked. Deafness is rare. Vision is never directly impaired. Taste may be affected. Giddiness is sometimes severe. Respiration may be irregular and abnormal in acute lesions, and the action of the heart irregular. Hyperpyrexia may result from acute lesions, even from hæmorrhage, which elsewhere, at first, depresses the temperature. Pyschical disturbance is much more frequent than might be expected, and is perhaps produced by the damage to the fibres and gray matter which, as we have seen, connect the cerebellum with the frontal and temporo-occipital regions of the brain. It varies much in character.

The most vulnerable part of the *medulla oblongata* is the region occupied by the nerve-nuclei at and below the apex of the fourth ventricle; and the most characteristic symptoms are those resulting from the damage to these nuclei. It is rare for an acute lesion to cause symptoms in the limbs, because a small lesion rarely occupies the pyramidal lesion, and any other than a very small lesion usually causes rapid death by interference with the cardiac and respiratory centres. Hence, also, if limb-symptoms do occur, they are slight; they may be unilateral or bilateral. A small tumor may, however, paralyze the limbs on one side, and the hypoglossal nerve on the other; but these symptoms depend more frequently on disease outside the medulla, compressing it and the nerve-trunks. The spinal accessory and glosso-pharyngeal nerves may then suffer with the hypoglossal, causing the group of symptoms that I have already described (p. 91). Of acute lesions, hæmorrhage usually kills with great rapidity. Necrotic softening from disease of the vertebral, occluding its branches, sometimes causes symptoms of sudden onset in the muscles supplied by the bulbar nuclei. There is palsy of the lips, tongue, palate, pharynx, and sometimes of the larynx, constituting acute labio-glossal paralysis, or acute bulbar paralysis. The loss of power is usually equal in the muscles of both sides, the lesion being seated commonly near the middle line. The palsy is occasionally irregular in distribution. The onset may be attended by subjective sensations in the legs, tingling, formication, etc., and rarely by some motor weakness. Chronic degeneration of the nuclei causes a palsy of similar distribution, but differing in its course, being gradual in onset and progressive in course, and often associated with muscular atrophy elsewhere.

Active disease of the *middle peduncle of the cerebellum* causes very peculiar symptoms, the chief being an involuntary "forced" movement of the trunk on its longitudinal axis, sometimes toward, sometimes from the side of the lesion. It may amount only to an irresistible tendency to lie on one side. There is usually vertigo, and sometimes it is very intense. The eyes may be directed to one side even when there is no tendency to fall, or there may be a difference in their height, or nystagmus. These symptoms are absent in stationary lesions, and so probably depend on active irritation, and not on a loss of nerve-structures. Associated with these there are often other symptoms of disease of one side of the pons—paralysis of the fifth nerve and of the opposite limbs.

The *middle lobe of the cerebellum* seems to be concerned in some way with the maintenance of equilibrium, and its disease causes unsteadiness in standing and walking. The feet are placed wide apart, and the defective balance may cause the patient to progress in a zig-zag manner. Sometimes there is a tendency to fall forwards or backwards. The gait resembles that of a drunken man. There is not the irregular movement of the legs common in locomotor ataxy. A sense of giddiness often accompanies the unsteadiness, but is not its cause, since either may exist alone. The unsteadiness may be present when the lesion is stationary, as well as in active disease. Vomiting is also frequent, but occurs only when the morbid process is active. The intimate connection of the vagus with the nerve-structures concerned in equilibration is seen in the vomiting that attends severe vertigo, such as that from ear-disease, and in the curious phenomena of sea-sickness, which probably depend on oscillations of the endolymph in the semicircular canals. This connection probably underlies the vomiting from cerebral disease. The vomiting may occur apart from giddiness. The same combination of vomiting and giddiness occurs in labyrinthine vertigo, but the giddiness is more distinctly paroxysmal, and the vomiting occurs only in connection with the giddiness. Disease of the *hemispheres of the cerebellum,* away from the middle peduncle, causes *per se* no definite symptoms of diagnostic significance. As Nothnagel first pointed out, the unsteadiness of movement does not result from disease in the hemisphere unless it is of such a character as to compress the middle lobe, and it is on this compression that the symptom depends. Compressing lesions, such as a tumor, in either the middle lobe or hemisphere, exert pressure on the pons, the tentorium limiting the pressure to the subtentorial region. Hence there is often slight weakness of the limbs, with signs of descending degeneration, increased myotatic irritability, etc., and sometimes feelings of numbness and tingling, rarely anæsthesia. When the tumor is in the middle lobe of the cerebellum, the limbs suffer on both sides; when in one hemisphere, the limbs suffer chiefly on the opposite side. The cranial nerves arising from the pons are also sometimes compressed, and the sixth nerves suffer earlier and more than others, on account of their exposed course over the convex surface of the pons. The facial and auditory nerves sometimes suffer from lateral pressure. Convulsions are rare in disease of the cerebellum, but tonic spasm, tetanoid in character, may occur in tumors, and is probably due to pressure on the pons.

In the ventricles of the brain only two lesions are common, hæmorrhage and liquid effusions, and the localizing symptoms are subordinate to those special to the morbid process.

It may be well to recapitulate the chief facts that we have considered, looking at them from the side of the symptoms instead of localities.

Persistent hemiplegia of the ordinary type may be due to disease of the motor path anywhere above the middle of the pons, or to disease of the motor cortex; but transient hemiplegia only shows that the disease is near the motor path or centres, so that it can affect them indirectly. Since the most common seat of disease causing hemiplegia is the corpus striatum and internal capsule, there is always a presumption that simple hemiplegia is due to a lesion in this situation. If the face and tongue escape, the disease may be anywhere in the path between the medulla and cortex, but there is a presumption that it is above the internal capsule, where the elements of the motor path are wider apart, and partial escape is more probable. The same is true, *à fortiori,* of partial hemiplegia affecting face and tongue, or face and arm. These, if of sudden onset, are most frequently due to disease of the cortex, sometimes to that of the white substance, very rarely to a lower lesion. I say "if of sudden onset," because when a chronic lesion involves the motor path anywhere, the gradual impairment may be at first partial in extent; but if the path, at the seat of disease, occupies a small area, all parts soon suffer, and the initial weakness of one limb, as it deepens in degree, soon extends to the other parts of that side. Considerable loss of sensibility on the extremities of the limbs, not through the whole side, probably indicates a lesion of the cortex or white substance beneath. Well-marked hemianæsthesia with hemiplegia indicates an extensive lesion of the posterior limb of the internal capsule; and conjoined hemiopia indicates that the lesion is extensive at the posterior part of the capsule, and has damaged the optic path, probably at the corpus geniculatum, or optic thalamus. Affection of the other special senses has the same indication of extensive mischief in this situation, but it is more common to have these symptoms without than with persistent hemiplegia, because a lesion in this region does not often destroy the motor part of the capsule. Paralysis of a cranial nerve on the side opposite to the hemiplegia indicates a lesion at the level of origin of that cranial nerve—the crus in the case of the third nerve, the middle of the pons in that of the fifth, the lower third of the pons in the case of the facial or sixth, the junction of the pons and medulla in the case of the auditory, the medulla in the case of the hypoglossal or spinal accessory. It must be remembered, however, that the localizing significance of these associations depends, in the case of acute lesions, on their simultaneous onset.

Ptosis, slight in degree, on the side of the hemiplegia, indicates that the lesion is in or near the cortex, with a slight probability that it involves the parietal lobe, or subjacent white substance. Conjugate deviation of the eyes from the side paralyzed, or towards it if there are convulsions or much rigidity of limb, is of no localizing significance; towards the side paralyzed, without any indications of spasm, it suggests, but does not prove, an affection of the pons; if there is complete loss of power of the external rectus, and slighter loss of power of the opposite internal rectus, the lesion is certainly in the pons. General convulsions are of no localizing significance: those that begin locally, if they occur at the onset of an acute lesion, suggest, and, if they attend a chronic lesion, practically prove, that the disease is in, or just below, the cortex.

Paralysis of the cranial nerves has a far more definite significance when several nerves are affected, than when one suffers alone. The possible seats of the disease are numerous in the latter case, while in the former they are few, and often there is only one position in which a single lesion can cause the combined effect. We may glance at a few of the more important indications. Palsy of one hypoglossal is probably due to disease outside the medulla, and this is certain if the palate and vocal cord (spinal accessory) are affected also. Paralysis of both sides of the tongue is usually due to disease of the nuclei (very rarely to symmetrical disease of the cortex), and the lips usually suffer with the tongue. If the face is paralyzed, we must note first whether the lower part suffers chiefly, and the muscles of the forehead and eyebrow escape, and electrical excitability is normal, or whether all parts are involved, and faradaic irritability is lost. In the first case the lesion is above the nucleus, and is probably above the internal capsule. In the second it may be in the nucleus, the fibres of origin in the pons, at the base of the brain, or in the temporal bone. Paralysis of the lower part of the face and tongue on one side indicates disease of the hemisphere, and probably of the cortex. If the auditory nerve suffers with all parts of the facial, and there is no ear disease, the lesion is at the base. Paralysis of all parts of the face on each side is due to symmetrical disease of the facial nuclei, or of the nerve-trunks at the base.

Paralysis of one ocular nerve is usually due to disease at the base;

that of all the nerves of one eye indicate disease about the orbital fissure; that of all the nerves of both eyes, degeneration of the nuclei. Paralysis of the sixth and facial (without the auditory) is due to disease within the pons. Paralysis of the sixth and fifth indicates disease outside the pons; although the nerves arise at some distance from each other, the sixth nerve, as it enters the dura mater, is nearer the fifth than is any other nerve.

The diagnosis of the seat of disease at the base of the brain has to be made chiefly in the case of chronic and subacute lesions, especially tumor and meningitis. In hæmorrhage the local symptoms are rarely prominent—they are lost in the more diffuse symptoms that attend severe hæmorrhage. In all basal disease the characteric symptoms are due to the interference with the cranial nerves. In disease of the posterior fossa, the fifth and those below are liable to suffer, and with them the motor tract, causing weakness in the limbs, usually hemiplegic in character. The distinction from disease within the pons depends on the circumstances that the nerves more often suffer before the motor tract to the limbs when the lesion is at the base, and that the combination of nerves affected is different. Thus, at the base, the fifth and sixth nerves may suffer together, the facial and auditory, the spinal accessory and hypoglossal. The nerves are often irritated before they are paralyzed, and such irritation of the fifth nerve often causes very severe and persistent neuralgic pains in the regions supplied by it, and sometimes (although rarely) trophic changes in the eyeball. Rigidity in the limbs may occur from the same cause, especially when the anterior pyramids of the medulla are exposed. General convulsions are rather more common from disease outside, than from that within the pons. Pressure on the middle peduncle of the cerebellum may give rise to intense vertigo and forced movements.

In the middle fossa, symptoms are chiefly produced by tumors, and the most important are symptoms of irritation and paralysis of the fifth nerve. The Gasserian ganglion is often involved, and trophic changes in the eyeball are common. In the interpeduncular space, morbid processes affect one or both third nerves, and often also one or both motor tracts in the crus. Less commonly one optic tract is involved, causing hemiopia. Convulsions are rare.

In the anterior fossa, properly speaking, only the olfactory nerves suffer, but disease usually involves also the neighborhood of the sella

Turcica, and damages one or both optic nerves, and the nerves to the eyeball in the wall of the cavernous sinus or at the orbital fissure. Occasionally the optic chiasma is involved; the fibres that decussate suffer chiefly, causing temporal hemiopia.

LECTURE XV.

DIAGNOSIS OF THE NATURE OF THE LESION—PATHOLOGICAL FACTS BEARING ON DIAGNOSIS.

GENTLEMEN,—We pass now to the last diagnostic problem that we have to consider, that which is, beyond all question, the most important—the nature of the disease. The diagnosis of the seat of the lesion is a problem of fascinating interest to the physician, but the limits of its importance to the patient are quickly reached. It is important to the latter chiefly as affording, in some instances, a guide to prognosis. But on the nature of the lesion depend not only the prognosis for the most part, but the treatment altogether. This pathological diagnosis is, as a general problem, far more difficult than the anatomical diagnosis. It is true, the nature of the disease can sometimes be determined with ease and confidence; more often, however, it is a question of much difficulty and some uncertainty.

We may ask, in the first place, what are the morbid processes that we have to deal with? We may leave out of the question those that are of extreme rarity, and consider only those that you are likely, at some time or other, to meet with—that constitute about ninety-nine out of every hundred cases of organic brain-disease that come under observation. They are not very numerous, fortunately for us. They are inflammation, chiefly of the membranes, hæmorrhage, necrotic softening, tumors, aneurism, abscess. Congestion and anæmia of the brain, which are not, properly speaking, organic diseases, we shall have to notice incidentally, and likewise three degenerative diseases—the bulbar degeneration that I have already mentioned to you, termed, from its symptoms, "labio-glosso-laryngeal paralysis"; "general paralysis of the insane," which differs from other varieties of insanity in the obtrusive physical symptoms that form part of its manifestations, and often come into diagnostic relation to other diseases; and, lastly, "disseminated" or "insular sclerosis."

To understand many of the indications of these lesions, it is needful for you to know certain facts regarding their mode of production and pathology. It is impossible for me to enter, however briefly, into the pathology of all these lesions. But the diagnostic indications, that I shall have to describe to you, will be clearer if some facts, regarding especially the acute lesions, are fresh in your minds. It may be well, therefore, for me to depart, in this lecture, from the special subject of this course, in order to direct your attention to these points.

Although we shall deal with congestion of the brain only incidentally, you should know that doubt has been thrown on the very occurrence of this condition—doubt that is a reaction from another extreme. The state of the blood-vessels of the brain after death is very little indication of what their condition has been during life. Before this fact was known, post-mortem distention was regarded as evidence of ante-mortem congestion, and an extensive symptomatology was elaborated on insufficient grounds,—a symptomatology that has, to some extent, survived its data. Further, "congestion of the brain," like "congestion of the liver," is a ready diagnostic refuge, seductive in its simplicity, and pleasing in its preciseness; easily affirmed, and not easily disproved. On this basis of pure diagnostic fancy, as I hinted in a previous lecture, a symptomatology has also grown up, and even statistics have been amassed, the value of which is considerably below zero. It is not surprising that from such reasoning a reaction has arisen, and the influence of cerebral congestion in causing symptoms has been narrowed almost to the vanishing point. Even its pathological possibility has been denied, on an old ground that the total amount of blood within the cranio-vertebral cavity cannot vary because the cavity is a closed one. The truth probably lies between the two extremes. Cases in which the pronounced symptoms can reasonably be ascribed to active cerebral congestion are rare—far less frequent than might be imagined from the place that the condition still occupies in the written and unwritten pathology of the present day. But the state probably does occur, and does cause symptoms. The mobility of the cerebro-spinal fluid permits the condition of the vessels to alter in various parts of the brain. If the cranio-vertebral cavity were hermetically closed, the variations could, perhaps, be only relative, not absolute. But the numerous foramina of this cavity are occupied by structures of little resistance, which may yield in some degree. Moreover, the large surface-veins of the spinal cord, and still more the enormous plexuses outside the spinal dura-mater, must constitute an important means of adaptation. The processes of secretion and absorption of the cerebro-spinal fluid, always in constant operation, must be influenced by the degree of pressure, and may quickly vary with it. The enormous variations in the amount of blood in the nerve-centres after death may be admitted as affording some evidence that variations are possible during life. Lastly, the eye is closed at least as completely as the cranio-vertebral cavity, and variations in the amount of blood within it may be actually seen. If, then, you should hear the possibility of cerebral congestion denied, remember that the grounds on which it is denied are not themselves unimpeachable.

Cerebral hæmorrhage is almost always due to the rupture of an artery, very rarely to that of a vein. Veins rupture chiefly under such extreme pressure as, for instance, attends strangulation, and, under the same circumstances, capillaries may give way and cause minute extravasations. Arteries, however, rupture only when their walls are diseased, and they may then give way without any extraordinary pressure of the blood. Before they burst, their wall yields before the blood-pressure; becoming extended, it becomes thinned. This bulging of the wall constitutes an aneurism, and hence it is that the hæmorrhage always results from the rupture of an aneurism. A healthy wall never yields before the blood-pressure; local change, lessening the elasticity of the wall, always precedes dilatation. The aneurisms that result may be on the large arteries of the base or surface, and are then of some size; or they may be on the small arterial branches, especially on those that go to the central ganglia, and are then minute. They are often called "miliary aneurisms," from their small size. The larger aneurisms on the arterial trunks are meant when we speak of "intracranial aneurism," without the qualification of "miliary." They are often single, and always few. The miliary aneurisms are always numerous; sometimes there are hundreds scattered through the brain. The difference in name has its justification, not only in the difference in size, but also in the fact that the miliary aneurisms cause no symptoms until they rupture, while the larger aneurisms sometimes cause considerable disturbance, especially when they are seated on a vessel at the base of the brain. The local change in the wall that permits the vessel to yield before the pressure of the blood is usually syphilitic disease or inflammation of the wall—either traumatic inflammation, or more often that which results from imperfect obstruction by a plug that comes from an inflamed cardiac valve, and excites an inflammation in the wall of the vessel similar to that in its source. The aneurism sometimes results from simple fibroid degeneration, but rarely from the fatty degeneration that is termed atheroma. The miliary aneurisms are chiefly the effect of a primary degeneration of the wall, of which an important cause is the strain to which they are exposed, aided by the degenerative tendency that is incidental to age, and that attends certain constitutional diseases, especially kidney disease. Thus the agent in their rupture, the blood-pressure, is in some measure the agent of their production. The reason why they are so frequent on the branches to the central ganglia is probably because these small arteries come off directly from a large vessel, and so the blood-pressure within them is higher than it is in most arteries of the same size. Although the degeneration differs in nature from the atheroma on the larger arteries of the base, their causes are in part the same, and therefore the two often coincide. Indeed, it has been suggested by Nothnagel that the atheroma may aid the production of the minute aneurisms, by the loss of elasticity in the larger arteries that it involves; this renders the pulse-wave more sudden in the branches. From these facts it follows that, while there is no necessary relation between the two, atheroma is present in three-fourths of the cases in which there are miliary aneurisms (Charcot and Bouchard). It is important to remember this fact. Certain general diseases cause a tendency to hæmorrhage, apparently from an acute degeneration of the walls of the smaller arteries. These are scurvy, purpura, pernicious anæmia, and especially leucocythæmia. They may cause cerebral hæmorrhage. The actual rupture of an artery may be produced by the strain of some muscular effort, or it may occur when the patient is at perfect rest, even, indeed often, during sleep. The frequency of rupture during sleep is remarkable. Perhaps it is determined by the influence of gravitation; or rather by the loss, in the recumbent posture, of this aid to the return of blood from the head. Possibly, moreover, the contraction of the vessels, that is said to attend sleep, is of arteries smaller than those that are the seat of miliary aneurisms, and may even increase the blood-pressure in the latter, and help to determine rupture.

The most frequent seat of hæmorrhage is the corpus striatum, the vessels that rupture being especially the branches of the middle cerebral that pass through the lenticular nucleus to the caudate nucleus, or optic thalamus. The extravasation ploughs up the adjacent parts, and usually tears through the internal capsule, causing hemiplegia. Often it extends into the white substance of the hemisphere, or bursts into the lateral ventricle. Although hæmorrhages may occur in any part of the brain, they are comparatively rare in the cortex—a fact of considerable diagnostic importance. Meningeal hæmorrhage usually results from the rupture of an internal extravasation, or from traumatic causes, but it sometimes occurs in maladies that entail a hæmorrhagic tendency, and in the course of severe acute specific diseases. Ventricular hæmorrhage is also usually secondary, rarely primary.

The extravasation of blood destroys the tissue-elements by lacerating them, and it compresses the adjacent structures. All mechanical injury irritates nerve-elements at the moment of its occurrence, and the irritation that attends hæmorrhage is probably one agent in the production of the loss of consciousness that usually attends the onset. This is doubtless due also to the pressure to which the whole brain is exposed, striking evidence of which is afforded by the extent to which the falx is sometimes bulged to the other side. It is to this compression that the "indirect" symptoms are chiefly due. But the adjacent damage thus produced is not usually intense in degree, although it is wide in range, and the ultimate recovery from these indirect symptoms is often perfect.

The term "softening of the brain" is used in medicine in its literal sense. You have probably already discovered that the popular use of the word is somewhat metaphorical, and that it is applied to various conditions in which there is a slow failure of mental power—conditions which we call by other names, because there is no actual softening. The chief pathological causes of softening are inflammation and arrest of blood-supply. There is, indeed, a third form, simple chronic softening, but it is excessively rare (not more than half a dozen well-authenticated cases are on record), and we need not, therefore, concern ourselves with it. Inflammatory softening we may also leave for the present; it is rare, except as the result of injury (which is beyond our province), or as the first stage of abscess, and that we shall consider separately. The arrest of blood-supply is due to the obstruction of an artery, and causes a sudden cessation of all the processes of nutrition that maintain the vitality of the tissue-elements. It is, therefore, often called "necrotic softening." This arrest of blood-supply, whatever be its cause, always involves derangement of the collateral circulation, and considerable secondary inflammation about the necrosed area. Hence the extent of the damage that falls short of destruction is usually relatively greater than in hæmorrhage.

Two pathological processes may cause the occlusion of an artery. A plug, from some distant source, may be carried into the vessel by the blood, and be arrested where the artery is narrower than the plug—"embolism"—or the clot may be formed in the artery by coagulation of the blood at the spot obstructed—"thrombosis." Embolism is the result of morbid process elsewhere in the vascular system, commonly in the heart. Thrombosis is the result of a local disease of the artery, by which its calibre is narrowed and its inner surface is changed. The alteration in the wall of the vessel is usually the result of atheroma or of syphilitic disease. The process of occlusion is often aided by a change in the blood, rendering it more prone to coagulate, or by a slower movement of the blood, giving it more time to coagulate. These may not only aid arterial disease in causing the occlusion, but may also sometimes cause thrombosis when there is no change in the wall of the vessel.

Although the processes of thrombosis and embolism are thus distinct, and usually occur under different conditions, the two may be combined in a secondary manner, which does not, however, affect the important and essential distinction between them. If an artery is obstructed by embolism, the distal portion may be further occluded by coagulation of the stagnant blood. Again, if a clot forms in an artery, it may be detached, and may be moved a little further on. This is, *de facto*, embolism, although the result of thrombosis, and classed as thrombosis, since the clot is formed in the artery occluded, although not at the point of occlusion. Again—and this is very important,—under certain circumstances the influences that cause thrombosis may also cause embolism. A state of the blood, inclining it to coagulate, may cause a clot to form, not in an artery, but in the heart, especially when there is valvular disease, and the clot may be detached. We shall presently see the importance of this consideration.

In embolism the source of the plug is between the pulmonary capillaries and the artery obstructed, and it usually comes from the left side of the heart, from a diseased valve, or from a clot in one of the cavities of the heart (especially from the auricular appendix), rarely from the aorta or the lungs. The valvular disease that is most frequently the source of a plug is mitral constriction, perhaps because the slow flow during the diastole favors the collection of corpuscular vegetations on the valve, and the rapid flow during the systole of the auricle favors their detachment. When the endocarditis is septic, and the vegetations contain organisms, the obstructing plug is apt to excite similar inflammation in the brain; and if the endocarditis is virulently septic, or the plug comes from a septic focus in the lungs, the inflammation in the brain may go on to suppuration.

The arterial disease that causes thrombosis is chiefly, as I have said, atheroma or syphilitic disease. The latter occurs at all ages at which acquired syphilis is prevalent; it is rare from the inherited disease. The atheroma is essentially a senile change, but its occurrence is facilitated and hastened by Bright's disease and some other influences. It increases in frequency as life advances. Both atheroma and syphilitic disease of the arteries are sometimes symmetrical, and may thus give rise to symmetrical lesions in the brain, and symmetrical symptoms in the limbs. Remember that traumatic injury, such as a fall on the head, or a blow, may cause arterial disease, which may lead to thrombosis long after the injury, perhaps after it has been long forgotten.

Thrombosis may occur, not only in the arteries of the brain, but also in the cerebral sinuses and veins. That in the sinuses is an extremely grave affection, not only by reason of its effects, but also because it is generally due to disease that is in itself most grave, and of which the thrombosis forms only a terminal incident. Its causes are of two kinds. The first is general disease, favoring clotting by changing the quality and retarding the flow of the blood. This form occurs with especial frequency in young children, and occasionally in very old age. Now and then it occurs at the end of phthisis, after childbirth, or in the course of cancer. In the young, its most common cause is exhausting diarrhœa, or some other prostrating disease, such as an acute specific. The sinus affected in these cases is almost always the superior longitudinal. I drew your attention, in an early lecture (p. 36), to the conditions of the circulation in the veins and sinuses, that render a general cause so effective. The second great cause of thrombosis in the sinuses is adjacent disease.

Almost any sinus may be thus affected. The disease is often in the bones of the ear or of the skull; sometimes it is outside the skull, as erysipelas of the scalp. This form occurs with nearly equal frequency throughout life, but it is rare at those periods at which the primary thrombosis is most frequent—infancy, and extreme old age. Inflammation of the wall of the vessel usually precedes coagulation of the blood within it. You may remember that I drew your attention (p. 37) to the communications between the sinuses and the veins of bones and exterior of the skull, which permit the ready extension of morbid processss.

The clot that forms in a sinus often extends into the veins. The obstruction of a vein causes intense hyperæmia of the part from which the blood should be removed by the vein; the capillaries rupture, and the minute extravasations that result may occupy almost all the area of the affected region. Some softening results, but there is not the wholesale softening that is produced by arterial occlusion.

In text-books on Medicine you will find very little about thrombosis in the cerebral veins, as distinguished from that in the sinuses. The condition is rarely seen post-mortem, but it is now and then met with. I believe, however, that it is both a frequent and an important lesion, and I must digress for a few minutes to give you the grounds for my belief. The condition is rarely seen in the post-mortem room, because, if a patient dies, the clot extends into the sinus, and the case is regarded as one of thrombosis in a sinus. Thrombosis, limited to a vein, although it may be serious in its effect on the brain, is not a lesion that involves much danger to life. The patients recover, and we can only infer the occurrence of the lesion from the character of the symptoms. I have seen it post-mortem in phthisis, and it has been observed in other conditions of prostration. It is occasionally met with in the course of tubercular meningitis, and may, I believe, occur when there is meningeal tubercle, but no inflammation. When it has been met with as an isolated lesion, one or two large veins over one hemisphere have been found occluded. The symptoms that suggest its occurrence are most frequently met with in children who are suffering from debility, exhausting diseases, especially acute specific diseases, or after blows on the head. Now and then they occur in very young children without any exciting cause, and I have observed that such cases, like infantile spinal paralysis, frequently occur during the hot season of the year.

The symptoms are these:—young children are attacked with hemiplegia, and recover, usually with some persistent weakness in the arm, often with mobile spasm in the limbs,—"infantile hemiplegia," or "infantile spastic hemiplegia," the cases are termed. The onset is often attended with severe convulsions, and convulsions may recur, even through life, affecting chiefly the paralyzed limbs, and constituting a distinct variety of epilepsy.[1] We have already seen, in the lecture on the local diagnosis, that such symptoms indicate a cortical lesion. The conditions under which this hemiplegia comes on make it almost certain that the lesion is thrombosis. Thrombosis in arteries is a very rare post-mortem lesion in childhood, far more rare than is combined thrombosis in sinuses and veins. The initial symptoms in these cases resemble closely those that have been observed in the rare cases in which a vein of the cortex is occluded in an adult. When children die with such symptoms as I have mentioned, we commonly find thrombosis in the longitudinal sinus, and veins opening into it. As I have said, the thrombosis probably extends into the sinus, and into the veins of the other hemisphere, during the final period. From these considerations it seems to me practically certain that, in the cases that do not die, there is thrombosis in one of the surface veins that ascend to the longitudinal sinus, in which, for the reasons I give you, the circulation is so slow, and the conditions are so favorable to coagulation. When such cases die in later life, the appearances are in perfect harmony with this view. We do not find any cavity, such as we do find when an artery has been occluded by embolism in childhood, and the subject dies in adult life. We see that in a certain area of the cortex the convolutions are shrunken and indurated—exactly the state that we should expect from the appearances presented by recent cases, and very unlike that observed in cases in which we are sure that there has been occlusion of of an artery.

These cases are not at all infrequent. They constitute the majority of cases of sudden cerebral disease in childhood. The hemiplegia may be transient, when the lesion is near, but not in, the motor part of the cortex; and probably, in early infancy, the initial hemiplagia, if slight in degree, may altogether escape observation. Most cases of epilepsy dating from infancy, in which the fits are one-sided, are of this character. We can very often trace slight indications of hemiplegia, in trifling weakness, slight shortening of the limbs, or in a slight degree of the mobile spasm that is so marked in the more severe cases.

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[1] See "Epilepsy and other Chronic Convulsive Disorders," p. 127, for an analysis and description of this form.

There is another frequent form of infantile cerebral palsy about which you will also find little in your manuals. In this form, paralysis of one side, or more commonly of both sides, is what is called "congenital," that is, the child comes into the world with it. The affection, like most others of a congenital character, is popularly attributed to "maternal impressions," to some emotional disturbance that the mother endured during her pregnancy. The designation is not altogether inapt, for the actual mechanism is a maternal impression, although it is one in which the mother is the agent, and not the subject. The cause is the injury the child suffers during its passage into the world. The affections are most common in first children, and after lengthy labors, and they are more common when the presentation is unnatural than when the head presents. They occur in forceps cases also, but probably the effect is due much less to the instrument than to the conditions that render instrumental aid necessary. The chief pathological mechanism is meningeal hæmorrhage, compressing certain parts of the brain. In children who die, there is usually a thick layer of blood over part of the cortex. The clot often lies over the motor convolutions, and may extend from the longitudinal fissure to the fissure of Sylvius. There is sometimes extensive sub-tentorial hæmorrhage from laceration of the cerebellum, but the blood over the cerebrum seems generally to come from the meningeal vessels, and not from the brain-substance. The child often suffers from convulsions during the first few days of life, and the limbs may even then be observed to be motionless. After a time some movement returns, and the ultimate condition is a combination of palsy and spasm, with spontaneous movements, and an irregular choreoid inco-ordination, constituting what has been variously termed "double spastic hemiplegia," or "bilateral athetosis," or "congenital chorea." The irregular movements are chiefly marked in the arms; in the legs the spasm is chiefly tonic, extensor, but with occasional flexion of the knees, especially on an attempt to walk, and often there is some contracture of the calf-muscles. In many cases there is mental defect, which may amount to actual idiocy. The children do not begin to walk until late, often not until the age of six or eight, and their walk is always peculiar. Adductor spasm in the legs may cause a "cross-legged" progression. You may sometimes recognize the unfortunate subjects of this defect in the streets by their peculiar gait. In addition to these motor limb-symptoms, there are often symptoms of paralysis of the muscles of the neck. The children are unable to keep the head up during the first two or three years of life. I have observed this neck-palsy chiefly in cases of foot or breech presentation. We do not know whether it is due to injury to the bulbar nerves (as the spinal accessory), or to their compression by extravasated blood, or to damage from the inflammation excited by such extravasation. It is conceivable that the anterior pyramids of the medulla may be directly injured, and that this may cause some of the limb-symptoms; but this is on the whole improbable, since hæmorrhage over the cerebrum and about the medulla may coincide. The symptoms sometimes suggest that the inflammation about the medulla closes the openings into the fourth ventricle, and that internal hydrocephalus may thus be produced. Occasionally the legs are affected in much greater degree than the arms, perhaps because the hæmorrhage is situated over the upper part of the motor convolutions (leg-centre), or these may even be directly damaged when the edges of the sagittal suture greatly overlap. Such cases have been termed "congenital spastic paraplegia." Again, the tongue and lips may be weakened out of proportion to the other parts, and difficulty in deglutition and in speaking may persist through life. Some of you saw such a case a few days ago in my out-patient room. This bulbar palsy may be the result of damage to the bulbar nerves, such as I have just mentioned, or it may be due to symmetrical hæmorrhage over the lower parts of the motor convolutions, which may produce symptoms closely resembling those that are caused by disease of the medulla oblongata.[1]

Inflammation of the brain, except from injury, or in the form of abscess, is so rare, that we may leave it out of consideration. The chief form of intracranial inflammation is that of the membranes—meningitis. Inflammation of the dura mater is rare, except from injury; the variety of chief medical importance is the inflammation of the pia mater, or pia arachnoid, since both the softer membranes usually suffer together.

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[1] The relation of these symptoms to injury during birth was pointed out by Dr. Little in 1862. Some valuable facts relating to the question have been recently published by an American lady, Dr. McNutt ("American Journal of Medical Science," January, 1885).

Meningitis may be simple, purulent, tubercular, or syphilitic. It may affect chiefly the convexity, or chiefly the base, or may be still more partial. Simple meningitis is rare except from exposure to the sun, and then it is seated over the convexity of the brain; but it occurs in limited extent from local disease of various kinds, such as tumor, or softening that is situated near the surface. Purulent meningitis is due generally to suppuration elsewhere, either adjacent to the membranes, as in caries of the bones of the skull, or disease of the internal ear. Distant suppuration produces it chiefly by the mechanism of pyæmia. It may attend the septicæmia of the puerperal state. Occasionally it occurs in the course of acute general diseases, and in children it may develop when no condition can be discovered to which it can be secondary. Purulent meningitis is often general, affecting both the base and the convexity, and in children the spinal membranes also.

The most common form of meningeal inflammation is the tubercular. It occurs in childhood with especial frequency, generally in families in which there is some tubercular taint. In adults it occurs chiefly in those who are actually suffering from tubercular disease of the lungs, but in youths of both sexes it may be primary. The membranes of the base are usually affected far more than those over the convexity, and the most characteristic symptoms of the disease are due to the damage to the cranial nerves. Remember that tubercle of the membranes is not quite the same thing as tubercular meningitis. Tubercle may exist without inflammation, and probably always precedes inflammation, which may be excited by some influence that may seem to be the cause of the symptoms. If the membranes were previously healthy, the exciting cause would be without effect. Massive tubercle, constituting a tumor, may be attended with intercurrent tubercular meningitis, and mixed symptoms of a complex character may result.

Syphilitic meningitis is almost always chronic. It may involve the membranes of the base, or of the convexity, but is often partial in distribution. It consists of a combination of inflammation and growth, and often extends about an actual syphilitic tumor, irritating and damaging the adjacent structures. Remember that the nerves are often damaged by syphilitic disease when there is no actual meningitis. They may be affected by a syphilitic neuritis, an inflammation of the sheath of the nerve; or the sheath may be the seat of a syphilitic growth. Such growths on the nerves are sometimes symmetrical; the same cranial nerve may thus be paralyzed on each side.

Almost any kind of tumor may occur in the brain, but some, as fatty tumors, which are common elsewhere, are extremely rare in the brain, while others, as glioma, are almost confined to the cerebral tissue. The most common intracranial growths are tubercular and syphilitic; next comes glioma, then sarcoma, and then cancer. Other forms are rare. Tubercular tumors are most frequent during the first twenty years of life. Syphilitic growths occur chiefly during the period of active adult life, from twenty-five to fifty. The only influence that ever seems to *excite* the development of a tumor is injury; this more commonly causes a tumor springing from the membranes than one beginning in the brain-substance. Growths that spring from the dura mater usually compress the brain-tissue. Those that spring from the pia mater, or develop within the brain, may either compress or invade the cerebral substance. The characteristic of glioma is its invasive tendency. It develops in the interstitial tissue of the brain, and incloses and destroys the nerve-elements. It enlarges the part of the brain in which it occurs, and there is no sharp line of demarcation between the tumor and the brain-substance. On the other hand, most other tumors, and among them tubercular, syphilitic, and sarcomatous growths, do not invade the brain-tissue, in the strict sense of the word; it perishes before them, partly by the compression they exert, partly in consequence of the inflammatory irritation that they produce, and which is shown by the zone of softening that usually surrounds them. In each case, however, the functional impairment of the cerebral tissue does not always correspond to the apparent structural damage. The nerve-fibres that are compressed before the growth in the one case, or are included in the growth in the other case, may retain some functional power, and the symptoms of loss of function may be much less than might be anticipated from the position and the extent of the lesion. Again, the irritation that is caused may be greater in degree, and wider in range, than the size of the tumor would suggest. I mentioned this fact to you on a former occasion, and remind you of it now on account of its importance. Lastly, you should know that one form of tumor, glioma, is occasionally the seat of a sudden hæmorrhage.

Abscess of the brain is usually due to injury to the brain, to adjacent suppuration, especially bone disease, or to suppuration at a distance, especially in the lungs. Curiously, it is rare in general pyæmia. Ear disease is its most common cause. It occasionally results from disease of the orbit or of the nose. The abscess may be in any part of the brain. Caries of the middle ear usually causes abscess in the temporo-sphenoidal lobe, rarely in other adjacent lobes. In caries of the mastoid cells the abscess is often in the cerebellum. The symptoms produced are frequently slight in proportion to the size and position of the abscess, and it is common for the disease to run an almost latent course until acute symptoms arise. These are often due to rupture, which may occur into the ventricles, or into the membranes, and its symptoms are of extreme gravity, quickly fatal.

Did time permit, I might with advantage point out to you many other facts of etiology and pathology that aid us in our diagnostic effort. Some of them I may mention in the description of the method of diagnosis to which we shall pass in the next lecture. But there is one general law that I must mention before I leave this part of the subject.

Whatever be the morbid process that causes damage to the brain, the symptoms are due to the latter; and the changes in the nerve-elements are much the same, whatever be the nature of the morbid process that causes these changes. We do not recognize this in our mode of speaking of these diseases. We speak, for instance, of "syphilitic disease of the brain," but the damage to the nerve-elements is never syphilitic. The syphilitic disease is outside them, sometimes altogether away from them, and it causes in them simple processes of degeneration, etc., on which the symptoms depend. This is not a mere theoretical refinement; it is of great practical importance, especially in regard to prognosis, and occasionally important in diagnosis. We shall see that we sometimes look to the result of treatment—to the effect of the administration of "anti-syphilitic" drugs—to confirm our diagnosis. But such treatment has no direct influence whatever, so far as is known, upon the changes in the nerve-elements. We may lessen or remove the syphilitic lesion that has produced the changes in the nerve-structures, and then certain kinds of damage will pass away, especially that which results from pressure, as of a growth. Other kinds of damage cannot pass away, as, for instance, necrotic softening, due to syphilitic disease of the wall of the vessel. You may remove this disease, but you cannot restore the brain-tissue that is dead, and no more recovery is possible for it than in a corresponding case of embolism. Symptoms may pass away in each case, but only when they are "indirect" in nature; and "direct" symptoms are as lasting in one case as in the other. Hence, before we can take the absence of improvement under treatment as an indication that a disease is not syphilitic, we must consider whether the change in the nerve-elements is one from which recovery is possible when the cause is removed. Speaking generally, the more quickly the changes are produced (and the symptoms developed), the less are they influenced by treatment; and in sudden lesions, as a rule, no influence is possible. Neglect of this consideration often brings discredit upon medical men. I have seen many cases of sudden hemiplegia, due to syphilitic disease of vessels, in which the patients were assured that if they were properly treated they would certainly get well; and when, after several years, there was almost no improvement, they were naturally inclined to think lightly, and sometimes to speak strongly, of the medical man who had given a prognosis so erroneous.

LECTURE XVI.

DIAGNOSIS OF THE NATURE OF THE LESION (*Continued*): SUDDEN LESIONS.

GENTLEMEN,—We proceed to-day to consider the methods of reasoning by which we ascertain the nature of the lesion. It is possible for me to give you, in these lectures, only an outline of the subject; but an outline may enable you to understand the principles and method of diagnosis, and even fix in your minds the most important rules. In the case of diseases that vary so widely in their character as do those of the brain, the most essential thing is to have a firm hold of the *methods* of diagnosis. It is only thus that you can hope to be able to deal with the problems that will confront you. No two cases are alike, and you will often be at fault if you trust to rules alone, and do not understand the principles on which the rules are based.

In distinguishing between the several lesions, we are guided in the first instance by certain indications. The first is the mode of onset of the symptoms; this enables us to distinguish between certain groups of lesions. The second consists of the causal indications,—the presence or absence of the conditions under which certain lesions occur, or the evidence of a similar lesion elsewhere in the system. A third is the position of the disease: certain morbid processes are more common than others in certain parts of the brain. A fourth is the character of the symptoms themselves, which may vary somewhat in the same part of the brain, according to the nature of the disease: this variation is slight at the onset, and is chiefly available when the course of the disease can be ascertained or watched. Lastly, other symptoms of the morbid process outside the nervous system may indicate its nature,—such as the pyrexia that attends inflammation.

The first question, then, is the time occupied by the onset of the symptoms—whether the onset is sudden, acute, or chronic. By "sudden" I mean the development of the symptoms to a considerable degree in from a few minutes to a few hours; by "acute," an onset that occupies from a day to two or three weeks; by "chronic," one that occupies more than a month. We are thus able to form a rough classification of the lesions into three divisions—the first comprehending the vascular lesions; the second, the inflammatory lesions; the third, the chronic inflammations, the growths, and the degenerative lesions. Cases of intermediate course are met with, but they can usually be brought without difficulty into one or other of these types, to which it is convenient to limit ourselves, not only for the sake of simplicity, but because they include nineteen-twentieths of the cases of organic disease. In thus using the mode of onset as a means of classification, we must take care that it is the actual onset that we consider. We shall have also to notice certain exceptions to the rules laid down—exceptions that are, however, rather apparent than real.

We may consider, first, the group in which the symptoms are sudden in their onset, developing often in a few minutes, sometimes occupying a longer time—a few hours. Remember, as a useful working rule, that symptoms of sudden onset, due to an organic cause, indicate a vascular lesion. They indicate the rupture of a vessel or the obstruction of a vessel; the former, as we have seen, causing hæmorrhage, the latter softening. The exceptions to this rule are few. They include the transient paroxysmal symptoms, such as convulsions or pain, and also inhibitory palsy; but the last is very rare. Again, an abscess of the brain may cause sudden symptoms by its rupture. This is the most important exception to the rule that sudden symptoms indicate a vascular lesion; but even here there are often chronic symptoms before the sudden symptoms that attend the rupture. Moreover, the symptoms that result from the rupture of an abscess are more often acute than sudden, according to the definition that I gave you of the sense in which we use these words.

We will take, then, the symptoms, such as paralysis, loss of sensation, etc., the sudden onset of which justifies us in suspecting the occurrence of a vascular lesion—softening or hæmorrhage. We have not merely to decide between the two, but we have, if the lesion is softening, to determine its form, that is, the nature of the obstruction that has caused it. The softening may be from the occlusion of an artery or of a vein. I told you, in the last lecture, the pathological causes of the various forms of obstruction, but it may be well for me to put them before you again in the form of a table.

A. Hæmorrhage—from rupture.
B. Softening—from obstruction,
    (a) in an artery—from
        (1) local thrombosis, due to
            (*a*) atheroma,
            (*b*) syphilitic disease,
            (*c*) blood-state;
        (2) embolism,
    (b) in a vein—thrombosis.

The actual onset may be preceded by premonitory symptoms. The significance of the presence or absence of these is limited. They are confined to those lesions that result from considerable preceding vascular disease—to softening from atheroma and syphilitic disease of vessels; they are absent in softening from embolism and in simple thrombosis. In ordinary hæmorrhage, premonitory symptoms, as such, are also absent. The miliary aneurisms cause no symptoms until they burst. Any premonitory symptoms in hæmorrhage are due, not to the cause of the hæmorrhage, but to coexisting atheroma, and have no direct relation to the lesion. In syphilitic disease and atheromatous softening, the premonitory symptoms are transient weakness or tingling in the limbs subsequently paralyzed, due to the diminution of the blood-supply afterwards arrested; and in each case there may be headache. In larger aneurisms, previous symptoms are those of a pre-existing lesion, causing pressure on some structure at the base of the brain, and are not true premonitory symptoms. The presence of premonitory symptoms may be of diagnostic importance, but their absence is of very little significance, since there may be none even in the diseases that are most often attended by them.

The actual onset is the next element in the diagnosis. The indication that it affords must ordinarily be used in subordination to the causal indications. Nevertheless it is important to consider it first, because it does occasionally afford a very strong indication, so decided as to override every other consideration. The chief guiding point is the effect on consciousness—the occurrence of apoplexy, its degree and duration.

Either softening or hæmorrhage may cause apoplexy, but it is produced by hæmorrhage more readily than by softening. Deep coma, lasting many days, is very rarely caused by vascular obstruction. On the other hand, it is not common for there to be no loss of consciousness in hæmorrhage, unless the extravasation is very small in size. Hence the absence of this symptom is not of much weight unless there is reason to think that the lesion is of large size. All that can be said is, that if there is complete hemiplegia lasting for some days, and no unconsciousness at the onset, the lesion is more likely to be softening than hæmorrhage; and if there is deep and prolonged coma, the lesion is almost certainly hæmorrhage, unless the patient is very old, and then there is some probability of softening, even with prolonged coma. If focal symptoms are slight, and initial apoplexy was well marked, hæmorrhage is much more likely than softening.

When the onset occupies several hours, the symptoms developing gradually without initial loss of consciousness, we must wait for the termination of the onset. If it ends in deep coma (ingravescent apoplexy), there is almost certainly hæmorrhage. If it ends in a stationary condition, without any loss of consciousness, the lesion is probably softening.

We must consider, next, the causal indications. They should not be taken in subordination to the mode of onset, but independently. The two should then be compared, their indications balanced, and their relative weight estimated. The first causal indication is the age of the patient. Hæmorrhage is so rare under forty, that it would not be suspected unless deep coma strongly suggested it. Softening from atheroma is still more rare under forty. This renders the diagnostic problem far more simple during the first half of life, since there remain only three common vascular lesions—embolism, thrombosis from syphilitic disease, and thrombosis from blood-states. We may conveniently, therefore, limit ourselves, in the first instance, to the cases that occur during this period of life. In each case we must begin by searching carefully for any indications of the presence of one of these three causes, always remembering, however, that hæmorrhage, though not probable, is possible, even in childhood.

In the last lecture we considered the chief causes of these lesions, and I told you that embolism, in the vast majority of cases, is from the heart, and is associated with valvular disease. The first point to be ascertained is therefore the presence or absence of such disease. In recent cases you are only justified in suspecting embolism, in the absence of valvular disease, when there is some other condition which may be the source of a plug (such as an aneurism of the aorta). But if the diagnosis has to be made a year or more after the onset of the hemiplegia, we cannot exclude embolism because we find no indications of valvular disease of the heart, if the onset occurred during, or soon after, such an illness as is usually, or occasionally, attended by endocarditis—acute rheumatism, chorea, or scarlet fever. I have known, for instance, mitral endocarditis during chorea to cause cerebral embolism, and a year later the heart had so far recovered that no evidence of valvular disease could be recognized.

There is one exception to the rule that valvular disease in a young person makes it probable that the lesion is embolism. Valvular disease may lead to cerebral hæmorrhage by causing an aneurism. I told you in the last lecture that the imperfect obstruction of an artery by embolism is one of the most common causes of intracranial aneurism. Hence, if the initial apoplexy is deep and prolonged, or if there is the "ingravescent apoplexy" already mentioned, the presence of heart disease not only does not exclude hæmorrhage, but increases its probability. This is true, even in children.

In thrombosis from blood-states alone, the causal indication is usually obtrusive. In adults this influence is almost confined to the post-puerperal condition. The clot may be formed *in situ*, or, if there is valvular disease, it may be formed in the heart, and carried to the brain. In other conditions favoring coagulation, such as phthisis and cancer, the clot generally forms in a vein. This is true also of the thrombosis that occurs so readily in young children, of which I spoke to you in the last lecture. The fact that the patient is very young and does not suffer from heart disease, or that the attack occurs during a state of considerable prostration, justifies a diagnosis of thrombosis; and if the symptoms are those of a cortical lesion, you may feel confident that the thrombosis is in a vein.

Thrombosis in an artery, from syphilitic disease of the wall of the vessel, occurs from six months to twelve years, or even more, after the primary disorder. In many cases there is a history of syphilis, and no other cause of arterial obstruction can be traced. We may then feel sure that a sudden cerebral lesion is due to this cause. If, however, we have no history of syphilis—as, for instance, in the case of a patient who is unconscious, and unable to give us his history—we have to rely on the indication afforded by the age, and on the absence of other causes. Often we are aided by the presence of premonitory symptoms, such as headache, or slight symptoms in the limbs that are afterwards paralyzed. These symptoms are of especial diagnostic importance in cases in which there is heart disease, and the patient has had syphilis. We then have to decide between embolism and syphilitic thrombosis. In the former, as we have seen, premonitory symptoms are absent. Hence, in such a case, any symptoms that suggest that there was a morbid process at work within the cranium before the occurrence of the vascular obstruction, make it probable that this obstruction is due to thrombosis, and not to embolism. For instance, a man who had old-standing aortic regurgitation was seized with hemiplegia. He had suffered from severe headache for a few weeks before the attack. Many years before, he had suffered from constitutional syphilis. The headache that preceded the hemiplegia could not be explained on the supposition that the lesion was embolism, and made it probable that the obstruction was due to thrombosis from syphilitic disease of an artery. Although there was no opportunity of ascertaining the exact nature of his lesion, yet it is probable that the diagnosis was correct, for a few weeks later some nodes appeared upon the skull, showing that the syphilis was still active in his system.

Syphilis, as well as heart disease, may cause cerebral hæmorrhage, and by the same mechanism, by causing an aneurism. Hence, also, a history of syphilis does not lessen the probability of hæmorrhage, if this is suggested by the character of the symptoms.

In the second half of life, the diagnostic difficulty is far greater. The causes operative during the first half continue during the second (although they become less effective as life advances), and, in addition, the potent mechanisms of arterial degeneration come into operation. Thrombosis from atheroma, and hæmorrhage, increase in frequency up to extreme senility; then, thrombotic softening becomes more frequent than hæmorrhage, and is more probable if other indications are equally balanced.

The most important indications in this period, are those drawn from the state of the vascular system. Arterial tension and a strongly acting heart suggest hæmorrhage; a soft pulse and an irregular, feeble heart suggest softening. If these indications are strongly marked, their significance is very great. Mere arterial degeneration tells us little. It tells us that the cause of softening exists—atheroma; but this so often coexists with the miliary aneurisms that cause hæmorrhage, that its significance is small. All that can be said is, that if other indications are equally balanced, considerable degeneration in the accessible arteries renders cerebral softening rather more probable than cerebral hæmorrhage. As we have seen, while atheroma is almost invariable in senile softening, it is absent in about a quarter of the cases of hæmorrhage. Advanced Bright's disease causes both atheroma and miliary aneurisms. It may thus be a cause of either softening or hæmorrhage. The former is, however, a rather more frequent consequence than the latter, perhaps because the miliary aneurisms attain an effective degree earlier than does atheroma, unless the patient has reached the period of senile degeneration. Hence, if other indications are equal, and the patient is not very old, Bright's disease suggests hæmorrhage; but if the other symptoms are such as to suggest softening, their significance is not appreciably lessened by the presence of Bright's disease.

As I have said, the difficulty in the diagnosis during the second half of life is increased by the circumstance that the vascular lesions common in earlier life—embolism, and thrombosis from syphilitic disease—occur during this later period. Thrombosis from the state of the blood and circulation also occurs, but the powerful influence of the puerperal state is no longer effective, and the influence of the circulation in causing thrombosis becomes to a large extent merged in that of the arterial degeneration, since it is rarely effective alone. On account of the frequency of the senile lesions, the mere presence of a cause of one of the earlier lesions,—a history of syphilis, or a source of embolism,—is of far less diagnostic significance than it is during the first half of adult life. The diagnosis rests, therefore, in larger degree on other indications than those of a present cause. If such other indications are absent, the diagnosis is a matter of probability only, or rather, I should say, of probability that is never high. All diagnosis that is founded on reasoning, and not on simple observation, is a matter of probability. Where several and different indications coexist, the degree of probability is often very low. The indications that guide us correctly in two cases may fail us in the third. A woman of fifty-five died with symptoms of a vascular lesion. The age of the patient made it probable that the lesion was either hæmorrhage, or softening from atheroma. Of the two, the symptoms suggested the latter. But the patient also had considerable mitral obstructive disease, and this made embolism a possible lesion. After death there was found, as expected, softening and not hæmorrhage; but the softening was due to syphilitic disease, extensive and characteristic, of the likelihood of which we had no suggestion from the patient's history. Dr. Hughlings-Jackson has recorded a case in which atheroma and syphilitic disease coexisted, and softening that had occurred was due only to the former. These cases illustrate the difficulty in diagnosis that results from the persistence during the senile period of the causes of vascular disease that are operative during the first half of life; and in such the diagnosis must usually be a matter of probability only, and of very low probability, and a conclusion, rightly reached, may unavoidably be wrong.

The second element in the causal indication is the evidence of a lesion elsewhere, of the same nature as that within the brain—evidence that a morbid process is at work in the system, such as may be the cause of the cerebral lesion. Most of the vascular lesions may occur outside the brain, and we may sometimes detect them. If they occur at the same time, or about the same time, as the cerebral lesion, they afford strong evidence of the nature of the latter. The occurrence elsewhere of the process of embolism may be recognized; as, for instance, in the spleen, by the enlargement and tenderness of the organ. The central artery of the retina may be obstructed by embolism, thus affording evidence of this process in the arterial system to which the cerebral vessel belongs. If, as I have seen, the retinal artery is occluded at the same moment as the cerebral artery, the evidence of the nature of the lesion in the brain amounts almost to demonstration. In syphilis we rarely have a more special indication of the precise process at work in the brain. Neither syphilitic disease of vessels, nor thrombosis therefrom, is to be recognized elsewhere, except in the rarest cases; but other indications that the syphilitic virus is still active may furnish equivalent evidence. Simple thrombosis, from blood-state, is occasionally accompanied by a similar accident in some other part, especially during the puerperal period. A clot may form in a vein of the leg, or some other part. Arterial or venous thrombosis elsewhere, from combined arterial disease and feeble circulation, coincides not unfrequently with cerebral thrombosis, and affords presumptive evidence of the nature of the lesion of the brain. Instances are senile gangrene of the foot, thrombosis in the veins of a leg or of the retina in those who are old and gouty, or feeble and depressed. Hæmorrhage elsewhere is rare in cases of cerebral hæmorrhage, except in the eye, and even there is significant only when the extravasation is large. The small hæmorrhages in the retina, that are so common in albuminuria, do not signify more than that the kidneys are diseased, and afford alone no more evidence that the cerebral lesion is hæmorrhage than does the renal affection itself. The significance of this we considered before. A large intra-ocular hæmorrhage, such as one into the vitreous, makes it highly probable that the cerebral lesion is of the same nature. But extravasations elsewhere, in the course of a general disease that is attended with a hæmorrhagic tendency, such as pernicious anæmia or leucocythæmia, also afford strong reason for believing that a cerebral lesion is of the same nature.

The position of the lesion occasionally helps us. A lesion of the pons, and still more of the medulla, that is not quickly fatal, is much more likely to be softening than hæmorrhage. The basilar artery is a frequent seat of syphilitic disease, and if other indications are equally balanced, in a case in which the symptoms suggest an obstruction of the basilar, as, for instance, the presence of heart disease and a history of syphilis, the fact that the basilar is the affected artery would turn the scale in favor of syphilitic thrombosis, against embolism. If the symptoms point to a cortical lesion, and other indications are equally balanced, this is much more likely to be softening than hæmorrhage. The sudden extension of the symptoms from one side to both, with renewed coma, that is produced by the rupture of an extravasation into the ventricles, constitutes evidence that the lesion is hæmorrhage, and not softening; but this significance is only decided when the extension occurs within a few days of the onset. Even then it is not a certain indication. Identical symptoms may attend the formation of a clot in a large artery of the other side of the brain in cases of softening, and if such symptoms come on after the first few days, it is even more likely that they are due to a fresh lesion than to the extension of the old one, and they therefore no longer have a definite significance. Hence it also follows that when there is decided reason to believe that the original lesion was softening, the occurrence of symptoms like those of ventricular hæmorrhage does not materially weaken the original diagnosis.

The character of the symptoms, in relation to the position of the lesion, does not give us much help in the case of acute lesions. Convulsions at the onset are of little significance. Those that succeed the onset, or occur during the subsequent course of the disease, are in favor of the lesion being softening, rather than hæmorrhage. An intense degree of secondary irritation is in favor of softening. So also are the athetoid movements, "mobile spasm," that may come on after the hemiplegia has existed for some months.

Here, for to-day, gentlemen, we may conveniently stop. There remains for consideration the diagnostic significance of the symptoms that attend the acute and the chronic lesions. But the chief acute lesion, according to the classification that I gave you at the outset, is meningitis. I propose, after we have completed our survey of the chief diagnostic indications from the side of the symptoms, to review them from the side of the lesions. It will be convenient to postpone the consideration of the symptoms of meningitis until we come to it in that survey. The symptoms that may result from chronic lesions we will proceed with at our next meeting.

LECTURE XVII.

DIAGNOSIS OF THE NATURE (*Continued*): CHRONIC LESIONS—SYMPTOMS IN THE SEVERAL MORBID PROCESSES.

GENTLEMEN,—The chronic lesions of the brain are chronic meningitis, tumor, aneurism, abscess, disseminated sclerosis; and we may include among them labio-glossal paralysis and general paralysis of the insane. In all these diseases well-marked symptoms develop, as a rule, slowly, occupying at least several weeks, and often several months, in their progress to a considerable degree of intensity. It is true that one of them (tumor) occasionally, and two of them (abscess and aneurism) frequently, have a latent course, and then give rise to acute and even sudden symptoms. But as pathological processes, they are chronic, and the symptoms of their development, when these exist, are correspondingly chronic in their evolution.

The first point to be considered in the diagnosis of these diseases is the causal indication that can be discovered. This element is rarely of much significance; it is far less important than it is in the sudden lesions of the brain.

The age of the patient may afford some guidance, since general paralysis, chronic meningitis, and aneurism are practically confined to adult life; aneurism and disseminated sclerosis being sometimes met with in youth. Tumor and abscess occur in all ages. Sex helps us only in so far as general paralysis is rare in females. Heart disease is a cause of aneurism alone among the chronic lesions. Syphilis may cause tumor, chronic meningitis, or aneurism. It is by far the most frequent cause of cerebral tumor in adult life. Chronic meningitis is very rare as a solitary lesion, and is due usually either to injury, chronic alcoholism, or to syphilis. In the absence of these causes it is very improbable. Injuries to the head may also cause abscess, or, in rare cases, tumor. The most common other causes of abscess are adjacent bone-disease, especially in the ear, and suppuration elsewhere, especially in the lung. If these are not present, abscess is unlikely. Tumors are usually primary.

and independent of growths elsewhere. Cancer is occasionally secondary, and hence a malignant tumor in some other part of the body renders it highly probable that symptoms of organic disease of the brain are due to a tumor. Tubercular growths in the brain are also occasionally secondary to tubercular disease elsewhere, but are more often primary, occurring in a subject predisposed by inheritance to tubercle. Hence either hereditary predisposition or actual tuberculosis suggests that a chronic disease is a tumor.

Neurotic heredity, indicated by a history of such diseases as epilepsy, insanity, neuralgia, etc., is rather against than in favor of actual organic disease of the brain. Equivocal symptoms are more likely to be due to functional disease. Preceding anxiety, mental shock, also render such disturbance more likely than coarse organic disease.

The diagnosis of the chronic lesions of the brain depends chiefly on the symptoms they produce. You doubtless remember the distinction between the diffuse and the focal symptoms. The diffuse symptoms, if any are present, are of much greater importance in the pathological diagnosis of chronic disease than are the focal symptoms, and we may, therefore, consider them first. If the symptoms are chiefly focal, we have to depend on their mode of development, and on their conformity to certain types. An instance of the latter is the case of labio-glossal paralysis, in which diffuse symptoms are entirely absent, but the focal symptoms correspond closely in all cases.

Of the diffuse symptoms we may consider, first, pain in the head. Headache is a conspicuous symptom in chronic meningitis, in tumor, and often in abscess. It is absent in the purely degenerative diseases. In aneurism it is sometimes marked, sometimes it is trifling. Occasionally it is absent. Remember that the characteristics of the pain of organic disease are severity and constancy, and the association with other symptoms. One of these is vomiting, which occurs in the same lesions as headache, and often coincides with the more intense paroxysms of pain. Of even greater significance is the association with optic neuritis. Rare in aneurism, optic neuritis is frequent in abscess and chronic meningitis, and most frequent and intense in tumor, in which it occurs, at some period, in five-sixths of the cases. If there is no cause of abscess to be discovered, persistent headache, and considerable optic neuritis, may be regarded as almost certain indications that the organic disease is a tumor. But remember that the optic neuritis of tumor is not always intense. In cases of slowly growing tumor, it may be slight in degree, and extremely chronic in course. Atrophy of the optic nerves varies in its significance according to its form. "Consecutive atrophy," that which follows neuritis, has the same significance as the neuritis that precedes it. Simple atrophy is rare except in the degenerative diseases, disseminated sclerosis and general paralysis of the insane. Its significance is always that of a degenerative process. Atrophy, that *follows* loss of sight, is due to damage to the optic nerve, and indicates either a tumor pressing on the nerve, or inflammation involving it. Loss of the reflex action of the pupil to light, without loss of sight, has the same significance as primary atrophy; it is due to degeneration, and indicates that a degenerative process is at work. It affords a ground for suspecting that other symptoms are due to a similar degeneration. It does not prove this. A coarse disease may coincide with a degenerative process. Such coincidence is now and then observed in syphilis, which predisposes to degenerations even during the stage in which it still causes organic lesions. Nystagmus occurs in tumor and in some degenerative diseases, but is not common in either class, except in disseminated sclerosis; in this it is a very frequent symptom.

Mental change may have various meanings, according to its form. Exaggerated delusions occur chiefly in general paralysis of the insane, but the early stage and slighter form of this disease are often attended by a simple optimism, in which, without any false idea, all things are looked at in a favorable light; the patient is happy under depressing circumstances, and is always "better" or "well" when physical weakness is steadily increasing. An unnatural complacency, without actual optimism, is also common in disseminated sclerosis. It is often marked, even in the early stage, and is a significant symptom that should always attract attention. This form of mental change is confined to these degenerative diseases, and is not met with in other organic lesions. Chronic delirium, sometimes active but amenable, may occur in chronic meningitis and tumor; occasionally the delirious condition resembles closely that of chronic insanity. More common in these diseases is simple mental failure—loss of memory, and slowness of speech—deepening to lethargy. In a considerable degree of this condition, fæces and urine are often passed without notice, although there is no paralysis of the sphincters. I have more than once mentioned to you the importance of this symptom.

Convulsions are absent in disseminated sclerosis, and also in most cases of aneurism, until rupture occurs. They are common in tumor and in chronic syphilitic meningitis, but not in alcoholic meningitis. They occur also, although not very frequently, in general paralysis of the insane. In each disease in which they occur, the convulsion may be general or partial, the latter form being, as you know, a focal symptom. Slight partial fits, recurring with great frequency, are almost confined to tumor.

Certain focal symptoms need special consideration, because they characterize certain forms of cerebral disease. Defect of articulation is produced by any disease of the medulla and pons, but it is also an early and characteristic symptom in the degenerative diseases—sclerosis, general paralysis, and labio-glossal paralysis. In the latter the defect is dependent on actual loss of power in the lips and tongue, which can be recognized as soon as there is much impairment of articulation; but in the two former diseases, sclerosis and general paralysis, there is at first no distinct weakness. The alteration in the early stage may be very similar in the two—a tendency to clip words, to run together syllables, which are thus confluent, instead of being "articulated." As sclerosis advances, however, there is usually a tendency to a separation of syllables. The staccato and elisive defect may coexist (see p. 92). In general paralysis, speech usually becomes hesitating, and is interfered with by manifest tremulous twitching of the muscles of the lips and face; often words are drawled out, and sometimes a guttural noise accompanies inspiration.

Another important group of symptoms are those of the eyeball muscles. Any disease at the base of the brain may paralyze these muscles in one or both eyes, but a slow progressive palsy, involving many muscles of both eyes, without other indications of basal disease, shows a progressive degeneration of the nuclei of the nerves, analogous to that of the bulbar nerves in the labio-glossal palsy.

Of the focal symptoms in the limbs, only one class is of sufficient pathological significance to need mention. Tremor is common in general paralysis, but may occur in tumor, and in chronic meningitis. Jerky inco-ordination of movement, accompanying weakness, is a characteristic symptom of disseminated sclerosis, but it occurs also in cases of tumor, chiefly in children (see p. 57).

We may now review these facts from a different side—from the side of the lesions,—and consider the aggregate of symptoms which indicate the several lesions, *i.e.*, the symptoms that these lesions produce in typical cases. In doing so I shall have to repeat many things that I have already said. I need not apologize for this, because, gentlemen, you have failed to learn the most important lesson in method of study if you object to repetition. You can only learn thoroughly by going over facts many times in their different relations.

We will commence again with the cases in which the onset is sudden, and take first the conditions in which there is loss of consciousness, apoplexy, but in which there are no indications of a one-sided lesion. There may be no symptoms on either side of the body and limbs, or there may be symptoms on both sides. These symptoms are in the state of the muscles, relaxation or contracture, or in the state of reflex action. We may thus divide these cases into two classes, according to the absence or presence of symptoms of bilateral character.

We will take first the cases in which there are no peripheral symptoms recognizable on either side. A patient may suddenly become unconscious, and remain so, and may not only present no indications of damage to one side of the brain, but there may be such restless automatic movements of the limbs on both sides, as indicate with certainty that there is no damage to the motor tract of either side. In most cases of the kind the coma is incomplete—there is stupor rather than coma. The patient, usually between forty and sixty, is of plethoric aspect, the pulse full, the carotids pulsate strongly, the face is flushed, reflex action is normal, the pupils are of medium size and act to light. In the course of two or three days the symptoms pass away entirely, and no indication of any focal lesion can be found, when consciousness has returned, and a thorough investigation is possible. Such an attack is probably due to cerebral congestion. It is customary to ascribe it to this cause, and the balance of evidence is in favor of the correctness of this opinion. The patient often has more than one attack of this character.

In the second class of cases the apoplexy is accompanied by evidence of interference with the motor centres on both sides of the brain. There is either complete relaxation of the muscles, or bilateral rigidity, or rigidity in some parts and resolution in others. In these cases we have to deal with a lesion either in or outside both hemispheres or in the pons. There may be ventricular hæmorrhage, meningeal hæmorrhage, obstruction of vessels in both hemispheres, hæmorrhage into the pons, or softening of the pons. If universal resolution and deep coma succeed, after a few hours or a day or two, the following symptoms—mental excitement, rigidity, convulsive movements (now in one part, now in another), and sudden headache—there is probably meningeal hæmorrhage, and this is especially probable if the symptoms succeed an injury. If, in a patient under forty, with heart disease or old syphilis, the coma is complete from the first, the limbs relaxed, and the symptoms continue for some hours without diminution, a cerebral aneurism has probably burst. Headache, giddiness, or palsy of cranial nerves for some weeks or months before the onset, makes this diagnosis still more probable. If initial coma is accompanied by indications of a one-sided lesion, rigidity or resolution of the limbs of one side, deviation of the head and eyes, followed in a few hours or a day or two by similar symptoms on the other side, without special affection of the cranial nerves, but with deep coma, the lesion indicated is ventricular hæmorrhage. If the symptoms are from the first bilateral, if there is rigidity or resolution, or the two are associated in different parts, if there are convulsive movements in both arms or both legs, interference with respiration within two or three hours of the onset, strong contraction of the pupils persisting or yielding to wide dilatation, a rise of temperature to 102° or more within an hour of the onset, the indication is hæmorrhage into the pons. If, however, similar symptoms come on gradually in the course of one or two days, the coma at first incomplete, and slowly deepening, without any early rise in temperature, with irregular affection of the cranial nerves, recognizable before the coma becomes deep, the indication is an occlusion of the basilar artery. Whether this is due to embolism or thrombosis must depend on the more sudden onset in the former, and on the causal indications. In the one case a source of embolism will be found, and perhaps indications of embolism elsewhere. In the other case these are absent: syphilis may be probable or certain; or the patient is in the degenerative period, with atheromatous arteries and a weak heart.

We will take next the case in which a patient is seized with apoplexy, and there is distinct evidence of a one-sided lesion—relaxation of muscles, loss of the skin reflex, change (loss or early excess) of the muscle-reflex action, deviation of the head and eyes. You are sure that a vascular lesion has occurred, on account of the suddenness of the onset; and unless the patient is a child, or is suffering from phthisis or other cause of extreme weakness, you are sure that it is an arterial lesion—an artery has given way, or become stopped up. For further guidance you examine the pulse, arteries, heart, and urine. At any age, after childhood, deep coma, lasting many hours, renders hæmorrhage probable, the more so if the patient is over thirty-five, and is suffering from Bright's disease. The significance of prolonged coma as an indication of hæmorrhage is much greater in early and middle life than it is in old age, since in the latter it is produced by arterial occlusion much more readily than in the former. Mind, I am speaking of actual coma, and not of the mere hiatus in conscious memory, which the patient afterwards describes as unconsciousness. He may tell you of a period of unconsciousness lasting for many days, when what we term manifestations of consciousness returned in a few hours. The indications afforded by the prodromata, onset, pulse, arteries, and heart, I have already described, and need not here repeat, beyond reminding you that embolism is suggested by valvular disease of the heart, especially during the first forty years of life, by a sudden onset without prodromata, by the brevity or absence of initial loss of consciousness, and by the evidence of embolism elsewhere. Syphilitic disease is suggested by a history of syphilis, or failing this, by its possibility combined with the absence of other causes, by the occurrence of prodromata (headache, symptoms in the limbs afterwards paralyzed), by an onset that is sudden or deliberate, but without, or with only brief, loss of consciousness. Hæmorrhage is suggested by the degenerative period of life,—but is not absolutely excluded even by youth; by deep and prolonged coma,—but is not excluded by brief coma, or even by the absence of any loss of consciousness. It is suggested also by high tension of pulse, a strongly acting or hypertrophied heart, the absence of prodromata, an initial fall of temperature, and the presence of Bright's disease. Softening from atheromatous thrombosis is suggested by the degenerative period of life, and, unlike hæmorrhage, is excluded by youth or early adult age. It is further suggested by a dilated, feeble, and especially an irregular heart, by previous slight attacks of the same nature, by prodromata in the limbs afterwards paralyzed, by the brevity of initial coma, and *à fortiori* by the absence of loss of consciousness at the onset.

Simple arterial thrombosis, from the blood-state alone, may be suspected if there is a constitutional state known to favor thrombosis, especially the puerperal state, and if no source of embolism can be discovered, and no cause of arterial disease can be traced.

Venous thrombosis is suggested by previous profound prostration and weakness, and especially by the patient being an adult in the last stage of phthisis, or a child under five years of age, and by the occurrence at the onset of convulsions in, or beginning in, the limbs afterwards paralyzed,—convulsions that indicate a cortical lesion.

Sinus-thrombosis is indicated by somnolence increasing to coma, and attended by general convulsions, coming on in a young child prostrated by diarrhœa or some other exhausting disease; or in a patient who has external disease adjacent to a sinus. In the latter case, focal symptoms may be present, which vary according to the sinus occluded. The diagnosis is only certain, however, when external tumefaction near the position of the sinus succeeds the other symptoms.

The indications that I have given you hold good, not only of hemiplegia, but also of other symptoms of an organic lesion, hemianæsthesia, hemiopia, and the like, which cannot, as a rule, be recognized until initial loss of consciousness has passed away.

We may now pass to the chief acute lesion of the brain—meningitis. The symptoms that should lead you to suspect meningitis are the combination of headache, vomiting without gastric cause, pyrexia, and delirium coming on in an acute manner. Remember that the absence of any one of these is of little negative significance. Headache is, however, seldom absent, and perhaps is never absent at all periods of the case, but is sometimes only trifling at the onset. On the other hand, it is usually severe, and the leading symptom. Remember, also, that the presence of only one of these symptoms is of no significance, and that this is true also of the combination of two of them—delirium and pyrexia,—which, without other indications of brain mischief, suggest a general and not a cerebral disease. The addition of moderate optic neuritis, of inequality of pupil, strabismus, palsy of cranial nerves, however slight, rigidity or weakness of limbs, retraction of head, or convulsions, adds very much to the probability of the diagnosis.

The diagnosis of the nature of meningitis is often less easy than the recognition of its existence. A family history of phthisis, or the presence, in an adult, of actual lung disease, suggests the tubercular nature of the inflammation, as, indeed, does the mere circumstance of childhood or youth, in which tubercular meningitis is far more common than any other variety. The absence of a cause of another form of inflammation is also an important negative element in the diagnosis. The indications of the other forms of meningitis are chiefly causal. Purulent inflammation may be suspected if the symptoms of meningitis follow suppuration elsewhere, near or distant, or signs of general septicæmia. The causes of purulent meningitis and of cerebral abscess are for the most part the same, and when an abscess causes acute symptoms, the diagnosis between the two is often difficult. It depends chiefly on the greater affection of the cranial nerves in meningitis, and on the history of previous more chronic cerebral symptoms in abscess. But it must be remembered that an abscess and purulent meningitis not unfrequently coexist.

In this outline of the most important symptoms of the sudden and acute lesions, I have said nothing of anæmia of the brain, because it is extremely rare for symptoms due to this cause to come into any diagnostic problem. Almost the only cases in which such symptoms are important are those of young children who are profoundly exhausted by diarrhœa. The patient may become somnolent and comatose; may sometimes present convergent stabismus, and even rigidity of the neck. These symptoms may pass away; or the coma may deepen, the pupils dilate, and the child die—the brain after death presenting no morbid change. The state was called "hydrocephaloid" by Marshall Hall from its resemblance to meningitis, which is sometimes called "acute hydrocephalus." The diagnosis from meningitis depends on the absence of focal symptoms and on the circumstances under which the symptoms came on. The distinction from sinus-thrombosis is still more difficult, and depends chiefly on the depression of the fontanelle, the absence of external tumefaction, and of limb-symptoms. Often, however, it is necessary to wait before a confident opinion can be given.

LECTURE XVIII.

DIAGNOSIS OF THE NATURE OF THE LESION (*Continued*): SYMPTOMS PRODUCED BY CHRONIC LESIONS, TUMOR, ANEURISM, ABSCESS, DEGENERATIVE DISEASES—DIAGNOSIS BETWEEN FUNCTIONAL AND ORGANIC DISEASES—CONCLUSION.

GENTLEMEN,—In the last lecture we commenced a survey of the groups of symptoms that indicate special lesions of the brain. We considered those that characterize the chief morbid processes that are sudden and acute. We pass now to the symptoms that attend the chronic lesions of the brain—chronic meningitis, tumor, aneurism, abscess, and the degenerative processes that we decided to include in our survey.

Chronic meningitis need not detain us long. The alcoholic form occupies the convexity, and causes diffuse symptoms, especially headache and delirium. Occasionally there is slight optic neuritis. It is probable that the inflammatory changes found in the membranes are part of a slight general encephalitis, rather than the actual cause of the symptoms. Syphilitic meningitis differs from all other forms in being local and never general. Hence its symptoms closely resemble those of a syphilitic growth. A positive diagnosis between the two is rarely possible, and indeed they frequently coexist. The chief difference is, that in meningitis the symptoms of irritation are greater than is the evidence of destruction, and the symptoms indicate a wider extent of mischief, especially at the base, than a growth would be likely to produce.

The symptoms that suggest the existence of a tumor are severe and persistent headache, vomiting, and optic neuritis, with progressive symptoms of interference with the functions of some part of the brain. These must be searched for, in every case, most carefully. Many of the most important are unnoticed by the patient. The pupils should be compared, their action to light ascertained. The movement of the eyes in every direction should be observed, and if there seems to be any defect, a colored glass should be used to examine for double vision.

The hearing should be tested, the strength of the masseters felt, the movements of the tongue and palate, and the closure of the vocal cords should be tested by making the patient cough. The voluntary, emotional, and associated movements of the face should be carefully observed. In the limbs and trunk the reflex action should be especially examined. A loss of the superficial reflexes in limb and trunk is sometimes the first objective symptom of tumor on one side. If there is evidence of the existence of a tumor, the next question is, Is the growth in and invading the brain, or outside the brain and compressing it? Optic neuritis is produced more readily by tumors in than by those outside the brain. If the symptoms indicate a growth of some size at the posterior part of the base, or over the convexity, and there is no optic neuritis, this is in favor of the growth being outside the brain. If the symptoms are those of a tumor of the pons, and the cranial nerves suffer before the limbs, and on one side before those on the other, the tumor is probably outside the pons, springing from the membranes in the posterior fossa of the skull.

There still remains one of the most difficult problems in cerebral diagnosis. What is the nature of the tumor? The answer to this question can sometimes be given with a high degree of probability, now and then with certainty. Often, however, the probability is so low that the answer is hardly more than a guess. The chief indications are as follows:—(1) The presence of morbid growths elsewhere, the nature of which can be determined. If the symptoms succeed a cancer or sarcoma elsewhere, it is practically certain that the intracranial growth is of the same nature. In rare cases the presence of an hydatid tumor elsewhere justifies the conclusion that the growth in the brain is of the same character. (2) Evidence of a general disease, of which an intracranial growth is known to be an occasional manifestation. Such diseases are tubercle and syphilis. In adults, signs of phthisis usually precede a tubercular tumor of the brain; in children there may be no other present indication of tuberculosis. A family history of tubercular or scrofulous disease is usually to be obtained. A physical configuration such as often coexists with a tubercular tendency is also suggestive. The symptoms or history of syphilis, congenital or acquired, render it highly probable that a tumor is syphilitic. The absence of a history of constitutional syphilis, if the patient has had a chancre, should not receive too much weight, since secondary symptoms are often unrecognized, or even absent. Moreover, in an adult, the syphilitic nature of a growth cannot be excluded unless we can exclude the *possibility* of infection, since even the primary disease may have been unnoticed, as in many cases in which constitutional syphilis is patent. These diathetic indications afford a probability of the nature of the growth—often a very high probability, but no more than a probability, since a cerebral growth of other nature may coexist with either diathetic state. (3) The age of the patient affords a little help. If the patient is under fifteen, and presents no indication of inherited syphilis, the tumor is not a syphiloma. If the patient is an adult, and presents no indication of phthisis, the tumor is not likely to be tubercular. (4) The seat of disease may give some assistance. In the cerebellum or pons, a tumor is likely to be tubercle or glioma, or, also if in the pons, it may be syphilitic. A cortical tumor, with signs of irritation, is probably syphilitic or glioma. A tumor of the base is probably syphiloma or sarcoma. A tumor outside the brain-substance is probably a sarcoma. (5) The course of the growth may be suggestive. A very slowly growing tumor is not likely to be syphilitic. A tumor that grows rapidly at the onset, and then becomes stationary, is probably tubercular or syphilitic. The occurrence of an apoplectic seizure of moderate severity, with sudden symptoms, is rare except in glioma. (6) Evidence that arrest and retrocession of the growth follow the administration of iodide of potassium or mercury is strongly in favor of the syphilitic nature of the tumor. If these drugs are without influence, and arrest follows a tonic treatment, the growth is probably tubercular. (7) Lastly, tubercle, glioma, and syphiloma are the most common forms of growth in the substance of the brain. There is always an intrinsic probability, therefore, that a growth will be one of these, and this may be allowed weight in the absence of other indications. Neither the intercurrent meningitis nor multiplicity of growth is of diagnostic significance. By a careful comparison of these considerations a probable diagnosis can be made in a large number of cases—that is, a diagnosis which will turn out more often to be right than to be wrong.

The symptoms that indicate the presence of an abscess of the brain are, for the most part, the same as those of tumor—headache, vomiting, optic neuritis, mental dulness, and focal symptoms. The latter are absent, however, far more frequently in abscess than in tumor, on account of the great frequency with which the abscess occupies the temporo-sphenoidal lobe, and on account of the tolerance the nerve-elements exhibit to the pressure of a slowly increasing encapsuled collection of pus. The distinction depends on the course and associations of the cerebral symptoms, and on the causal indications. (1) Instead of the uniform progress of the symptoms of a growth, those of abscess are for a long time slight in what is termed the "latent stage." They then develop rapidly, in an acute manner, like meningitis, or even in a sudden manner, like hæmorrhage, distinguishable from these by the definite character of the slighter chronic symptoms that preceded. (2) The ophthalmoscope is often of great value, revealing, during the latent stage, or at the onset of the acute stage, an optic neuritis, which excludes alike a mere vascular lesion and a commencing meningitis. (3) The general symptoms that attend suppuration—fever and rigors—are often present. (4) A cause of abscess (ear disease, suppuration about the skull or elsewhere, or an injury) can be found in most cases. With no cause ascertainable, and an absolute latency, an abscess can be suspected only on the indication afforded by the ophthalmoscope. There is no other cerebral lesion in which the patient may plunge, in a few hours, from apparent health into imminent danger, and in which considerable optic neuritis is found at the onset of the acute symptoms, when there is no general disease to which the optic neuritis can be due.

The symptoms of a small tumor at the base of the brain, in the position of a large artery, occurring in a person who is past middle life, or who has heart disease or syphilis, and, in the latter case, not yielding to treatment, justify a suspicion of the existence of an aneurism. The suspicion can only be raised to absolute certainty by the presence of a rare sign, a murmur audible on auscultation of the skull, practically met with only in aneurism of the internal carotid. The diagnosis of the precise artery affected depends on the local symptoms. I may mention a few of these indications, obtained by comparing the symptoms in many recorded cases. Optic neuritis is occasionally met with, but is not common unless the aneurism is adjacent to the optic nerve, *i.e.*, is of the internal carotid or anterior cerebral. The seat of pain is of significance only when occipital; it then points to the basilar as the artery affected. Loss of sight of one eye, sometimes extending to the other, with, or still more without, optic neuritis, with or without loss of smell on the side first affected, occurs in aneurism of the internal carotid and anterior cerebral. The distinction between the two depends on the occurrence in the former of paralysis of the motor nerves to the eye first affected. Paralysis of the third nerve without affection of sight, or with hemiopia, is produced by aneurism of the posterior communicating, and also, usually, with hemiplegia of the opposite side, by aneurism of the posterior cerebral. Affection of the fifth nerve alone is of little significance, but with bilateral weakness in the limbs, and difficulty of articulation or swallowing, it occurs in aneurism of the basilar. Paralysis of the cranial nerves below the sixth, associated with weakness of the limbs on one side, may be due to aneurism of the lower extremity of the basilar or of the vertebral, and symptoms of bulbar paralysis, of slow or sudden onset, may occur in the same cases. Slight hemiplegia is of little significance, but, if it is considerable, there is not likely to be an aneurism of either the anterior cerebral or posterior communicating artery. Simple general convulsions are very rare. If they begin locally, they suggest that the aneurism is of the middle cerebral artery, on the outer surface of the brain, and if they are opisthotonic, that it is of the basilar. All these symptoms are, of course, only significant in the presence of a cause of aneurism. The severe apoplectic symptoms which attend rupture increase much the probability of the diagnosis; but this is then a matter rather of scientific curiosity than of practical importance. Without preceding symptoms, the rupture of an aneurism may be suspected if sudden and deep apoplexy occurs in a person who has not yet reached the degenerative period of life, and has no renal disease, and especially if there is heart disease or a history of syphilis. If the apoplexy is attended first with unilateral and then with bilateral sypmptoms, the aneurism that has ruptured is probably of the middle cerebral. Symptoms of hæmorrhage into the pons suggest that the aneurism is of the basilar or of the posterior cerebral.

There remain the three degenerative diseases that stand, to some extent, apart from the other lesions of the brain—bulbar paralysis, disseminated sclerosis, and general paralysis of the insane in so far as its physical symptoms are concerned. An alteration in articulation is constantly an early symptom of the first, occasionally of the second, and frequently of the third. Tremor is a usual symptom of the two latter, but is absent in the former. In all, the symptoms are gradual in development, although chronic bulbar paralysis has its acute homologue, which depends on a sudden vascular lesion in the medulla. Although the resulting symptoms of the acute and chronic disease may be the same, the sudden onset takes the cases out of the present category of chronic lesions.

A defect in articulation, and afterwards in swallowing, depending on actual distinct loss of power in the parts concerned, is the distinctive characteristic of bulbar, or labio-glossal paralysis. I told you (p. 93) that the palsy is arranged around the tongue as a centre, affecting the lips, tongue, palate, pharynx, and often the larynx; and I described to you the symptoms that result. The ultimate aspect of the patient, with motionless tongue, open mouth, immobile lower face, and unmodulated phonation on an attempt to speak, is characteristic and unmistakable.

The distinctive symptoms of insular sclerosis are not specially cerebral. There are coarse jerkings in the arms, sometimes in the legs, occurring on movement only. They apparently depend on the development of islets of sclerosis in the motor tract, either within the brain or within the spinal cord, and are often followed by actual loss of power. Sometimes unsteadiness on the legs is one of the earliest symptoms. With these, however, are often associated symptoms that are distinctively cerebral. (1) A change in articulation, in which syllables are unduly marked off from one another, and even separated, and, at the same time, the endings of words are slurred. But with this there is no loss of power in the muscles concerned in articulation, at any rate until the latest stage of the disease. (2) Nystagmus. (3) A slight and inconstant degree of mental change. There may be at first some mental hebetude, but the most characteristic condition is an unnatural cheerfulness, and contentment with a state of disability which should naturally give rise to grave concern. The change never goes beyond this; but in this degree it is very common, and rarely fails to strike a medical observer as peculiar, although the friends regard it as merely the expression of an admirable resignation.

The early symptoms of general paralysis vary much. They may be mental or physical. The mental change that is most characteristic is the familiar "expansive delirium," as it is called, in which the patient revels in exaggerated personal delusions. Often, however, there is no more than a tendency to regard all things through rose-colored spectacles, without any actually false idea. There may not even be this optimism; there may be merely mental weakness and failure of memory. The physical symptoms are as much spinal as cerebral. Tremor on movement is often conspicuous; it interferes with the more delicate actions, and is especially conspicuous in the muscles of the lips and face in articulation. Speech is hesitating and drawling, with a tendency to slur word-endings. Loss of the light-reflex of the iris, and inequality of the pupils, are common. With these there may be no spinal symptoms, or there may be indications of either lateral or posterior sclerosis. Cases of what may be termed "pseudo-general paralysis," in which the characteristic physical symptoms exist almost alone—merely loss of memory and slight optimism representing the mental disturbance—are not uncommon, both in syphilitic subjects and in those who have not had syphilis. These cases run a much more benign course than does the classical form of the disease, often remaining stationary for years, and even improving. They do not get into asylums, and therefore are scarcely recognized in descriptions of the disease, although they are far from rare.

In conclusion, we may glance briefly at some of the more salient points in the all-important distinction between the so-called functional and organic diseases of the brain. The diseases that most frequently give rise to difficulty are hysteria, neuralgia, and some forms of epilepsy, but the chief diagnostic indications of the inorganic head-pain and convulsions have been already alluded to, and I need only now speak of the often perplexing symptoms of hysteria.

There are few organic diseases of the brain that the great mimetic neurosis may not simulate. Palsy and spasm, coma and convulsion, pain of every form and degree, giddiness, loss of sight, of hearing, of speech,—almost every symptom of positive lesion finds its counterpart in the repertory of that functional disturbance which lies, latent or manifest, within the potentialities of the nervous system of most women, and of many men, ready, in some, to spring into activity on the slightest touch of favoring circumstances. To lead you through the labyrinth of detailed distinction would occupy as many lectures as we have been able to devote to our entire subject. I must content myself with a briefer course—a course, indeed, that may perhaps be more useful to you,—and merely point out the general principles that must guide you. First remember this fact. Given the condition of age and sex—that is, of state of nervous system, developmental or other—that underlies hysteria, its manifestations may be evoked by any disturbance of nerve-function, whether this comes from without, as in the pure and primary disease, or from within, as in secondary hysteria, which may accompany almost any organic disease of the brain. I mentioned to you in a previous lecture how many organic maladies of the brain may evoke symptoms of hysteria. Therefore, given symptoms of hysteria, we must never infer that this is the primary disease until we have searched for, and excluded, the symptoms of organic disease. *A fortiori* this is true of the mere conditions in which hysteria occurs, and yet the diagnosis of hysteria is often made merely because the patient who presents symptoms of organic disease happens to be a girl. The slightest unequivocal symptom of organic disease is of absolute diagnostic significance, and until the absence of any symptoms of the kind has been ascertained, no other symptoms and no etiological circumstances should be permitted to bias the observer's mind. In a large number of cases, attention to this rule will dispel all difficulty. But there remain cases in which the only symptoms present are equivocal, and seem compatible with each disease, although not characteristic of either. This difficulty is usually rather apparent than real. The symptoms common to the two classes present differences of detail and grouping, and a thorough knowledge of the characters of organic disease enables the observer to discern these differences without difficulty. In the very rare cases in which the symptoms are absolutely equivocal, the history of other unequivocal symptoms of hysteria may be allowed weight and to turn the scale. For instance, a lad, after a period of excessive study, was suddenly seized with severe pain in the head, which lasted for a day or two, and then gave place to a state of stupor, in which he could only be partially roused to slowly swallow nourishment that was placed in his mouth. The condition resembled that of meningitis; but a similar condition is sometimes of purely functional origin. All other symptoms of organic disease were absent, and it was ascertained that the headache was preceded by a period of excessively frequent breathing (a characteristic hysterical symptom), which ceased suddenly when the headache came on. This justified a diagnosis of hysterical stupor, and two days after the onset the lad woke up free from any serious symptom.

It may be well to glance very briefly at the differences between some of these equivocal symptoms. Speaking generally, this hysterical character is suggested (1) by their onset after emotion, or after witnessing analogous symptoms in another. (2) By their increase on attention, and in the course of examination. (3) By their mutability; grave symptoms of one character will cease suddenly, and give place to others which could not result from the same organic cause as at the first. (4) By the differences between the symptoms of hysterical origin, and the corresponding symptoms of organic disease. For instance, we may take the one-sided motor palsy of hysteria—hemiplegia. There is rarely complete paralysis in both limbs, although there may be in one. The face is never affected—an important distinction. There is usually some contracture in the most paralyzed limb, and this, in the hand, involves usually the long flexor, flexing all the phalangeal joints (the metacarpo-phalangeal more than in late rigidity), and the flexion of the wrist does not relax the other joints as it does in organic disease. Moreover, all hysterical contractures present a distinguishing characteristic: when an attempt is made to overcome them, it is felt that the resistance is not uniform, but varies from moment to moment. When the paralysis is incomplete, movement is slow, and is attended by characteristic irregular tremor, far smaller in range than that of disseminated sclerosis, but coarser than simple tremor, and more irregular. If the muscles are felt or watched, it will be found that voluntary movement is interfered with by undue contractions in the opponents of the muscles that should effect the movement. There is no wasting, or change in electrical irritability, except after long disuse, and then only in trifling degree. The skin-reflexes are not lessened on the affected side; the knee-jerks are equal; there is no uniform foot-clonus (unless there is great contracture of the calf-muscles), but what I have termed a "spurious clonus" can occasionally be obtained, characterized by the palpable variations in the half-voluntary contraction of the calf-muscle on which it depends.[1] The onset of hemiplegia may be rapid, but it is rarely so suddenly complete as in organic disease, and is not attended by loss of consciousness. Anæsthesia is common, either one-sided and complete, or chiefly localized in the most paralyzed limb. Hemianæsthesia, coming on without other indications of a cerebral lesion, is almost always of hysterical origin.

The only derangement of the eyeball-movements that occurs in hysteria is convergent strabismus from muscular spasm, and it is easy to observe the absence of any paralysis if you make the patient, with one eye closed, move the other in various directions. Simulated ptosis is sometimes seen; it depends on a very gentle contraction of the orbicularis, that is transformed at once into a vigorous and demonstrative contraction, to keep the eyelid down, if the patient is made to look up with the other eye.

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[1] See "Diagnosis of Diseases of the Spinal Cord," 3d Ed., p. 32.

The well-known aphonia of hysteria, depending on under-action of the adductors of the vocal cords, is sufficiently distinguished by the absence of any attempt at phonation. This inactivity may extend to the tongue, and cause loss of even whispered speech, a sequence that is pathognomonic. Very rarely a palsy of the abductors may give rise to inspiratory stridor without impairment of expiratory phonation. The symptoms are those of the same palsy when of organic origin, but the association of the two is always sufficiently diagnostic. The convulsive attacks of hysteria ought not to cause a difficulty in diagnosis. These features are, for the most part, distinctive, and the problem is, not whether they indicate organic brain-disease, but whether any other symptoms of a different character coexist with them.

Here, gentlemen, we must end. The problems that we have considered in these lectures are certain to present themselves before you, frequently, in your future work. Their form will vary, but the principles of diagnosis that I have endeavored to put before you, will guide you, I believe, to a right conclusion in most cases. When you meet with special difficulty, do not be hasty in trying to arrive at a decision. Think over the symptoms; read over the description of the diseases between which the diagnosis lies; examine your patient again; and, if necessary, watch the symptoms for a time. Perplexing as these problems often are, they are rarely insuperable to those who combine, with a firm grasp of the methods of diagnosis, a fair knowledge of the symptoms of organic diseases of the brain.

THE
DIAGNOSIS OF DISEASES
OF THE
SPINAL CORD

THE

DIAGNOSIS OF DISEASES OF THE SPINAL CORD.

In the following pages an attempt is made to give an outline of the Symptoms and Diagnosis of Diseases of the Spinal Cord, with especial reference to those points on which modern investigation has added to the knowledge that is current in the profession. In order to make a description of these points useful, it is necessary to include them in a general outline of the subject, in which they may take their proper place.

Our knowledge of the symptoms of these diseases is in advance of our therapeutic power. But the study of diagnosis must not, on that account, be undervalued. Even for practical purposes our diagnostic knowledge needs to be ample and exact. A very superficial study of practical medicine will show that much diagnosis, which is of no direct avail for treatment, is essential for the diagnosis which enables us to treat successfully. Of all organs there are some diseases for which we can do little, there are others for which we can do much; but unless we are able accurately to distinguish the diseases of each class, we shall often fail to apply our skill where it would be effective. Moreover, the diagnosis is easy in some cases; while in others it is most difficult. The diagnostic knowledge which is superfluous in the one is essential in the other.

There is another reason why a general survey of the elements of the diagnosis of diseases of the spinal cord may be useful. In systematic treatises, types of disease are described. But the mutual relations of all parts of the nervous system are intricate, and its morbid states are correspondingly complex. Cases which conform to types are rare, and the untypical cases are often puzzling, and can only be understood by a clear conception of the general principles of diagnosis.

The first question in the diagnosis of diseases of the spinal cord is whether the symptoms are due to organic disease or to merely functional derangement. But although this is the first question in any case, the answer to it depends on the presence or absence of the signs of organic disease; this point in diagnosis cannot, therefore, be considered until we have discussed the character and significance of those signs. It is, however, important to note, at the outset, as a rule of cardinal importance, that the presence of a cause of functional derangement is not, in itself, sufficient ground for diagnosis. All signs of organic disease must be searched for and excluded, before the presence of the causes of functional disease is admitted as evidence. It is clear that, if there are any signs of organic disease, the existence of the causes of functional disease is of no significance whatever. Hence the importance of knowing accurately all the signs of organic disease, even those which are minute and may seem superfluous.

The causes of functional derangement frequently co-exist with organic disease. Hysterical symptoms, for instance, are often present in the subjects of organic disease in all parts of the nervous system. There are two reasons for this. Many organic diseases are the result of an inherited neuropathic disposition, which may also cause hysteria. Further, the damage from organic disease often affects very widely the nutrition and function of the nervous system, and thus leads to the manifestations of hysteria, in addition to the symptoms of the organic disease. Hysterical symptoms are often obtrusive, for instance, in cases of tumor of the brain. Hence the existence of such symptoms constitutes, alone, small evidence that a given disease is merely functional. It may seem superfluous to dwell upon so obvious a point, but I have often known cases to be regarded as purely hysterical, when the plainest signs of organic disease were to be found, if looked for; and this merely because the patient presented manifestations of hysteria. The same thing is true of other causes of functional derangement, and it is true of the simulation of disease. Circumstances suggestive of malingering should be allowed no weight until the signs of organic disease are proved to be absent. Not rarely the neglect of this obvious rule has led to cruel injustice. When we think that symptoms are simulated, we should always look on our diagnosis, as well as on the patient, with suspicion, and be very sure that we are right, before we act on our opinion.

If there is evidence of the existence of organic disease, we have to ascertain its seat and nature—to make, that is, the anatomical and pathological diagnosis. It is of importance to keep these two points distinct in our minds. Their confusion is a fertile source of error in diagnosis. It is true that certain parts of the nervous system are frequently the seat of certain morbid processes; but if we infer that because this or that region is diseased, the morbid process is of this or that character, we make a pathological diagnosis from anatomical facts; and such a diagnosis will, not rarely, be erroneous. It is true we have sometimes to use this mode of reasoning· in the absence of other evidence, or as corroborating other evidence, it is legitimate and useful; but it is only thus to be used, and always with full recognition of its character and uncertainty. As an instance, we may take the case of inco-ordination of movement of the legs—locomotor ataxy. This indicates disease of a certain part of the spinal cord. In the majority of cases the disease in this region is of a certain character; but in some, the symptoms being the same, the nature of the disease is different; and to infer the character of the morbid process, in the latter case, from the symptoms present, would lead us, not only to a wrong diagnosis, but to an erroneous prognosis and unwise treatment.

It is to be remembered, then, that we can only infer from the symptoms present in a case at a given time—the *seat* of the disease. To learn its *nature* we have to study the way in which the symptoms came on, and any associated conditions which may be present.

I have put this rule thus absolutely because it is one of great importance, often overlooked. There are, however, certain exceptions to it, especially the facts that pain, acute spasm, and sloughing of the skin are sometimes (not always) signs of an *irritative* lesion. Even here, however, the exception is rather apparent than real; for it is the acuteness of these symptoms, rather than their mere occurrence, which is of *pathological* import.

We will consider first, then, the elements of the anatomical diagnosis, the signs which indicate the seat of the disease—"localization," as it is the fashion to term it—and afterwards glance at the elements of the pathological diagnosis; that is, the symptoms which indicate the nature of the morbid process.

We can only learn the significance of symptoms by ascertaining their nature and origin—what they are and why they are. Hence our study of diagnosis must consist to a large extent in what may be termed symptomatic pathology. The symptoms of disease are alterations of healthy function, and much of our symptomatic pathology is, literally, perverted physiology. We must, accordingly, in the first place, have a clear conception of such points in the structure and normal function of the spinal cord, as may enable us to understand the origin of the symptoms of its diseases.

I.—MEDICAL ANATOMY OF THE SPINAL CORD.

The position of the cord, and of the origins of the nerves, in relation to the bony canal in which it lies, is the first important point which we have to consider. It will be remembered that the cord does not, in the adult, extend through the entire length of the spinal canal. It ends opposite the 1st lumbar vertebra, or opposite the interval between the 1st and 2d lumbar vertebræ. Hence the various pairs of nerves (except the highest) do not arise from the cord opposite the vertebræ at which they leave the canal, and after which they are named, but at a higher level. The difference between the level of origin and of exit, slight in the cervical region, increases as we descend the cord, until, as you know, in the cauda equina, the lowest nerves have a very long course from the end of the cord to their foramina. It is important to know what nerves correspond in their origin with a given part of the vertebral column, because the cord often suffers secondarily to disease or injury of the bones. The relation is rendered more complex by the fact that the vertebral spines, which alone we can feel, and which constitute, therefore, our localizing guides, do not correspond in all parts to their vertebræ. Since these points are important in diagnosis, and are not adequately described in any English work, I have prepared a diagram (Fig. 19) showing the average relations of the spines to the bodies of the vertebræ, and of both to the origins of the spinal nerves. The tips of the cervical spines correspond nearly to the lower borders of the corresponding vertebræ. Each of the upper three dorsal spines corresponds nearly to the upper border of the body of the vertebra below. From the 4th to the 8th dorsal, each spine corresponds to the middle of the body of the vertebra below. The 9th, 10th, and 11th spines slope less, and their tips again correspond to the upper borders of the next vertebræ, while the rest of the spines are opposite the bodies of their own vertebræ.

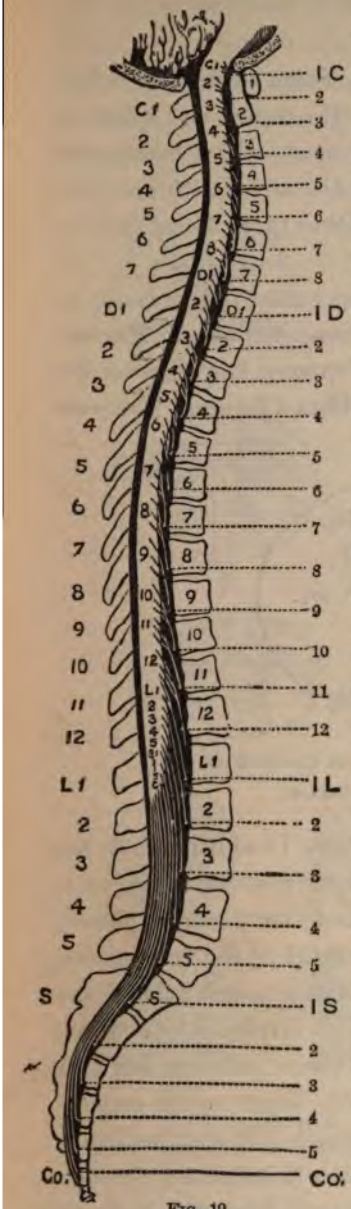

Fig. 19.

What is the relation of the spines to the nerve origins?[1] The first three cervical spines are opposite the origins of the 3d, 4th, and 5th cervical nerves. The 6th and 7th pairs arise opposite the intervals between the 4th and 5th, and the 5th and 6th, cervical spines respectively. The 6th cervical spine corresponds to the origin of the 8th cervical nerve, and the 7th cervical spine to the first dorsal nerve. The first four dorsal spines vary. The 1st spine corresponds to the interval between the 2d and 3d pairs, or to the origin of the 3d pair. The 2d spine is between the 3d and 4th pairs, or opposite the 4th pair. The 3d spine is opposite the 5th, or the interval between the 5th and 6th pairs. The 4th spine is opposite the lower part of the origin of the 6th pair, or even below it. The 5th spine always corresponds to the origin of the 7th pair; the 6th spine to the 8th pair; the 7th to the 9th pair; the 8th to the upper part of the 10th pair; the 9th to the 11th pair, and the 10th to the 12th pair. The 1st lumbar nerve arises opposite the 11th dorsal spine; the 2d lumbar opposite the interval between the 11th and 12th spines; the 3d and 4th opposite the 12th spine; the 5th dorsal and 1st sacral opposite the interval between the 12th dorsal and 1st lumbar spines, while the remaining sacral nerves arise nearly opposite the 1st lumbar spine.

I need not describe in detail the relations of the origins of the nerves to the bodies of the vertebræ, since they may be inferred from the facts I have given, or ascertained by a reference to the diagram.

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[1] The only recorded observations on this point are those of Nuhn and Jadelot. The facts stated in the text are partly the results of a fresh examination of the relations, kindly made for me by Mr. V. Horsley, Demonstrator of Anatomy in University College.

Thus the cervical enlargement of the cord, which ends at the origin of the 1st dorsal nerves, corresponds nearly to the bodies and spines of the cervical vertebræ, while the lumbar enlargement, which commences at the 12th dorsal nerves, corresponds to the bodies of the 11th and 12th dorsal and 1st lumbar vertebræ, and to the lower three dorsal and 1st lumbar spines.

We may next consider, briefly, the general structure of the cord, seen in a transverse section, such as is represented in the accompanying diagram (Fig. 20). It is divided into two halves by the anterior and posterior fissures *af.* and *pf.* The latter is rather a septum than a fissure. The position of each is marked by a depression in the surface. In addition there are two other depressions, one where the posterior nerve-roots enter (*pr.*); another (at *s.*) about midway between this and the posterior fissure. The two "fissures" do not meet, being separated by the commissure which connects the two halves. The gray matter, in each half of the cord, is surrounded by the white substance, and is divided into two portions, or "cornua." The anterior cornu (*ac.*) varies much in size and shape in different parts of the cord, being much larger in the cervical and lumbar enlargements than in the dorsal region. It does not come to the surface; the anterior nerve-roots (*ar.*) reach it by passing through the anterior column. The posterior cornu (*pc.*, Fig. 20) is much smaller, and comes almost up to the surface at the depression (*pr.*) where the posterior nerve-roots enter. It is much larger in the lumbar enlargement than in the cervical and dorsal regions. The white substance is composed of nerve-fibres running vertically; and since these

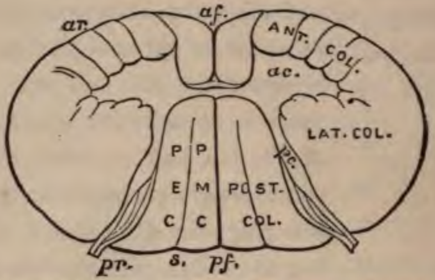

FIG. 20.—DIAGRAM OF SECTION OF SPINAL CORD IN THE CERVICAL REGION.
The reference letters are explained in the text.

end at different levels, the white substance lessens in amount from above down. The relative amount of the gray and white substance, and the differences in size and shape of the gray cornua in various regions of the cord, may be understood from the accompanying diagrams of sections at different parts.

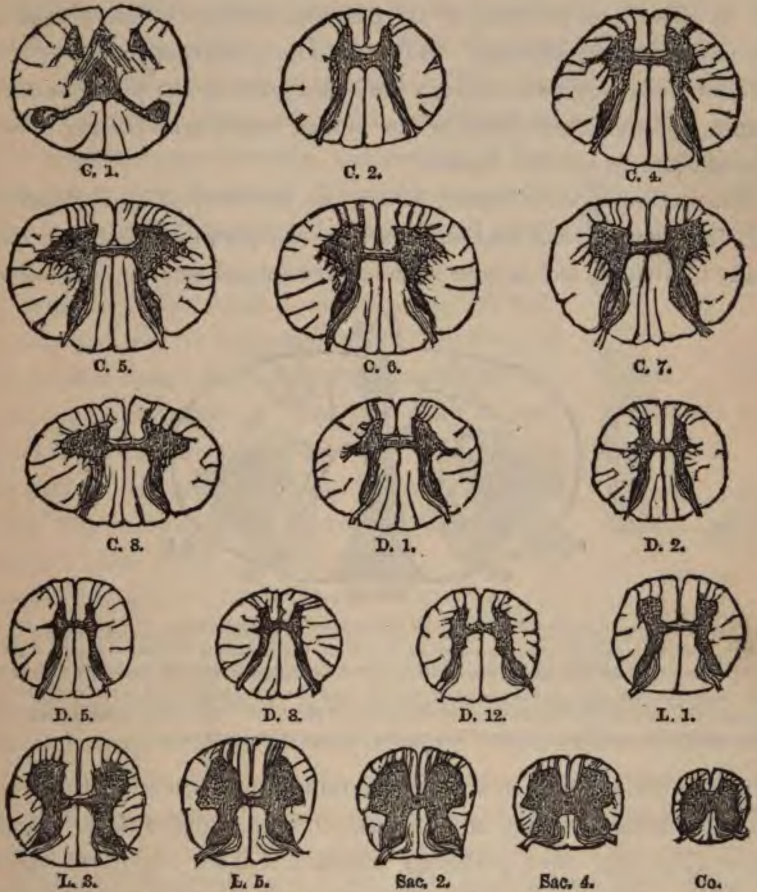

FIG. 21.—DIAGRAMS OF SECTIONS OF THE SPINAL CORD AT DIFFERENT LEVELS.

The letters and numbers indicate the spinal nerves to which the sections correspond. Each is figured twice the natural size. (From Quain's "Anatomy," 8th Edition.)

The posterior cornua, coming to the surface, cut off from the rest of the white substance, that which lies between them, and this constitutes the posterior columns. Each posterior column thus lies between the posterior median septum and the posterior cornu. The posterior roots of the nerves do not all immediately enter the gray substance, but some of them course through the outer part of the posterior column, which we may term the postero-external column (P.E.C., Fig. 20). It has been termed by Charcot the "posterior root-zone." A septum of connective tissue (*s.*, Fig. 20) separates off, from this area, that part of the posterior column which is adjacent to the posterior median fissure, and the part so marked off is termed the "posterior median column." The distinction of these two portions of the posterior column is, as we shall see, very important in pathology. But it is to be noted that the fibres of the posterior roots only pass through the outer part of the postero-external column, and that these fibres in the lumbar region pass further into the column than the cervical region.

The portion of white matter which lies in front of and outside the gray, from the anterior median fissure to the posterior cornu, is structurally undivided, and is termed the antero-lateral column. It has been artificially divided into an anterior column, lying to the front and inner side of the anterior cornu, and a lateral column, lying outside the gray matter (Fig. 20). But pathology indicates a more important division than this, and the study of the development of the cord corroborates the teachings of pathology. If certain parts of the brain (concerned in voluntary motion) are destroyed, certain fibres degenerate throughout the cord, and this degeneration marks out for us the fibres which are in direct connection with the motor region of the brain. Two tracts are thus picked out, one in the posterior part of the lateral column, on the opposite side to the cerebral lesion; and one on the same side, in the anterior column, close to the medium fissure (see Plate, Fig. 1, *a* and *b*). These

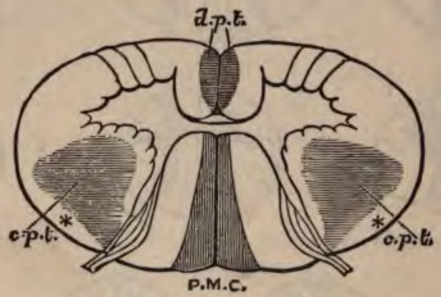

FIG. 22.—AREAS OF SECONDARY DEGENERATION.

P.M.C., posterior-median columns, one on each side of the posterior median septum; *d.p.t.*, direct, or anterior, pyramidal tracts, one on each side of the anterior median fissure; *c.p.t.*, crossed, or lateral, pyramidal tract in the posterior part of each lateral column, and separated from the surface of the cord by (*), the direct cerebellar tract of Flechsig. The areas of ascending degeneration are shaded vertically; those of descending degeneration transversely.

are called the "pyramidal tracts," because the connection of these tracts with the brain is by means of the anterior pyramids of the medulla. They are shown on both sides, shaded transversely, in Fig. 22. Those adjacent to the anterior median fissure are the "anterior (or direct) pyramidal tracts;" those in the lateral column are the "lateral (or crossed) pyramidal tracts."

The crossed (or lateral) pyramidal tract contains the motor fibres which have decussated in the medulla; the direct (or anterior) tract, those which have not decussated there. The relative size of these tracts varies in different individuals; the more fibres that have crossed in the medulla, the smaller is the direct tract, and *vice versâ*. The direct tract may even be absent, all the fibres having crossed above (Flechsig).

Regarding the lateral pyramidal tract, it may be observed that it is situated behind the level of the anterior cornu, that it does not usually extend quite up to the posterior cornu (although it may do so behind), and that it does not extend up to the surface of the cord, being limited by a zone (*Fig. 22), in which there is no descending degeneration. The fibres of this zone are said (by Flechsig) to descend from the cerebellum.[1]

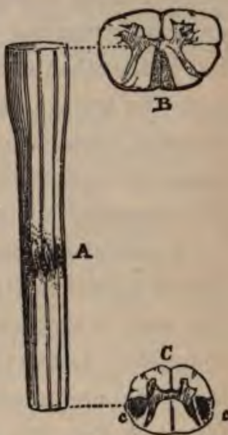

FIG. 23.

We have further evidence that the fibres in the posterior parts of the lateral columns descend from above, in the fact that, if the cord is destroyed at any level, these fibres on each side degenerate below the lesion, just as they do on one side after a cerebral lesion. Such bilateral degeneration is shown in Fig. 23, C, *c c*, the lesion causing it being indicated at A. Bilateral degeneration of these tracts is shown also on the Plate, Fig. 2,

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[1] The direct pyramidal tract is also called the column of Türck; the postero-median column is called the column of Goll, and the postero-external column is called the column of Burdach. I have avoided the use of these terms. This system of nomenclature is full of inconvenience, increasing the difficulties of the student, and leading to frequent mistakes in scientific writings. There are very few observations in medicine regarding which it is not obvious that they would speedily have been made by some one other than the actual observer; that it was very much of an accident that they were made by certain individuals. Scientific nomenclature should be itself scientific, not founded upon accidents. However anxious we may be to honor individuals, we have no right to do so at the expense of the convenience of all future generations of learners.

b b. This degeneration is currently, although not very happily, termed "sclerosis," and the degeneration of this area is designated "lateral sclerosis"—"descending lateral sclerosis," when it is the result of a lesion higher up.

I mentioned that the fibres of the white columns end at different levels, and so the white columns become progressively smaller. The portions of the white columns which constitute the pyramidal tracts follow the same rule, and hence the descending degeneration becomes smaller in area the lower we descend in the cord, and in the lowest part of the lumbar enlargement it is very small indeed. The fibres of the anterior pyramidal tracts disappear in the dorsal region, probably passing to the other side of the cord. Hence the descending degeneration from the brain, in the lower dorsal and lumbar region, is confined to the opposite lateral column. Hence, too, if the cord is compressed, the descending degeneration appears in the anterior pyramidal tracts only when the compression is high up.

A similar process of "secondary degeneration" furnishes corroboration of the distinctive division of the posterior column, which, as we have just seen, is suggested by anatomy. Below a point at which the cord is completely destroyed, although the lateral columns degenerate, the posterior columns present no change. Above the point destroyed, however, (as at B, Fig. 23), while the lateral columns, and the postero-external columns present no change, in the postero-median columns the nerve-fibres disappear and become replaced by connective tissue.[1] This ascending degeneration is also shown in the Plate, Fig. 3, *c*. These are the only secondary degenerations commonly described. But I have found in a spinal cord of which the lower extremity was crushed, a symmetrical area of slight ascending degeneration in the anterior part of the lateral columns, in front of the pyramidal tracts (Plate, Fig. 3, *e*). Of its possible significance I will speak presently.

The gray substance is composed of nerve-cells and interlacing fibres; some of the cells in the anterior cornua are very large and with many processes, and are called the "ganglionic" or "motor" nerve-cells. One process from each cell is undivided and constitutes the axis cylinder of a nerve fibre of an anterior roof. The other processes divide and subdivide in the substance of the gray matter.

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[1] This statement, although that which is current, is not strictly accurate. Some distance above the damage the ascending degeneration is confined to the postero-median columns; but close above the compression the degeneration extends outwards into the posterior portion of the postero-external column, not, however, to that part of it through which the posterior roots pass. Hence it is probable that the fibres which course upwards into the postero-median column enter it from the postero-external column.

II.—PHYSIOLOGY OF THE SPINAL CORD IN RELATION TO THE SYMPTOMS OF ITS DISEASES.

We may now consider the chief functions of the cord, and the effects of their impairment. In the spinal functions we have to distinguish two great systems of action—that by which the cord transmits and that by which it controls; *i.e.*, its functions as a conducting organ, and as a nerve-centre, reflex and automatic.

Motor Conduction.—The conduction of motor impulses from the brain is in the antero-lateral white columns, probably in the pyramidal tracts; it is chiefly in the side of the cord corresponding to the limbs moved, the crossing taking place for the most part in the medulla. The motor path leaves the cord by the anterior nerve-roots, but does not enter them directly. The fibres of the pyramidal tracts enter the substance of the gray matter, and are apparently connected through this with the large nerve-cells from which the anterior root-fibres proceed. Hence, both the matrix of gray matter and the motor nerve-cells form part of the motor path. The power of voluntary motion may be arrested by a lesion anywhere in this tract—lateral column of the cord, gray matter, and anterior nerve-roots. If the lesion is on one side of the cord, the loss of power will be on the same side, and in degree proportioned to the number of pyramidal fibres which have crossed in the medulla; and this, as we have seen, is not always the same.

Sensory Conduction.—All sensory impulses—of pain, touch, temperature—enter the cord by the posterior roots, passing, in part directly, in part through the postero-external columns, into the posterior cornu, and quickly crossing to the other side of the cord. There is some reason to believe that the paths of these several sensory impulses up the cord are not the same. That of pain has been commonly believed to pass up the central gray matter; that of touch, and perhaps also of temperature, passes up, in the opinion of some authorities, in the posterior column. But according to late, most careful, and apparently conclusive experiments by Woroschiloff (confirmed by Ott), such sensation as can be tested in the lower animals, is conducted in the dorsal region in the lateral columns. No facts have been hitherto recorded suggesting that this is true of man. But if sensation is conducted in part in the lateral columns, it is certainly not in that portion of them which is occupied by the pyramidal tracts, because there may be no loss when these are completely degenerated. It is probably, therefore, in front of these. This is the situation in which I have found the ascending degeneration in the case of crushed cord in which sensation was greatly impaired. (See p. 216, and Plate, Fig. 3, *e.*) This fact at present stands almost alone,[1] but, taken in conjunction with the experiments on animals, it points, I think, to the probability that some sensation is conducted in this region in man; what or whence, whether from the skin or deeper structures, we do not know.

We are still too ignorant of the paths of sensation for us to infer much from the form of its affection in spinal disease. One thing, however, seems clear—the path of sensation is less definite than that of motion. A very small portion of undestroyed cord will conduct sensation, but it is then, at least in its intenser form, commonly retarded. Each form of sensation may be impaired by disease of the posterior roots, either outside the cord or in the postero-external column through which they pass; or by disease of the conducting structures of the cord higher up; and since the paths decussate in the cord, if the lesion is unilateral, sensation will be affected on the side of the body opposite to the lesion (motion being affected on the same side). A strong reason for believing that the paths are not the same is that the senses of touch and pain and temperature are often impaired in different degrees. The most common change is for the sense of pain to be lost and touch preserved (analgesia). In such a condition the slightest touch of the finger may be felt readily, but a needle may be driven into the skin, and the patient experiences only the sensation of a touch. In other cases the sense of touch may be lost, and only the perception of painful impressions remain (anæsthesia). In other cases both are changed proportionately. To ascertain impairment, it is necessary to examine carefully the sensitiveness to each form of stimulation, to note how the patient feels the impression (since the sensation, when not lost, may be perverted), to note whether it is localized accurately, and to note whether it is unduly retarded. Sensations of pain and temperature are never so rapid as that of touch, and it is in these that the chief retardation takes place.

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[1] A confirmation of the occurrence of limited degeneration in this situation is furnished by an interesting observation by Dr. Haddon ("Path. Trans." vol. xxxiii., p. 21.

The functions of the ascending fibres of the postero-median columns are still unknown. Their degeneration does not seem to be accompanied by any impairment of sensation. We also know little of the function of those fibres of the antero-lateral column which lie in front of the anterior cornua.

Reflex Actions.—The next important function of the cord is its action as a reflex centre. We may regard the reflex system of the cord as made up of a series of nerve-loops, or arcs, each posterior, sensory, root being connected with certain anterior, motor, roots by means of the gray matter (Fig. 24). This consists partly of the large motor nerve-cells, and partly of a network of the finest nerve-filaments and minute nerve-cells. The connection of the roots, through the gray matter, is apparently by this network of fine fibres, which interlace like the filaments of a sponge. But in this there are paths of different resistance, so that a slight stimulus may pass by the most ready path to a certain anterior root, while a stronger stimulus may diffuse itself more widely and affect many nerve-roots. For instance, a gentle touch on the sole may cause only a movement of the toes; a stronger touch, a start of the whole leg. A

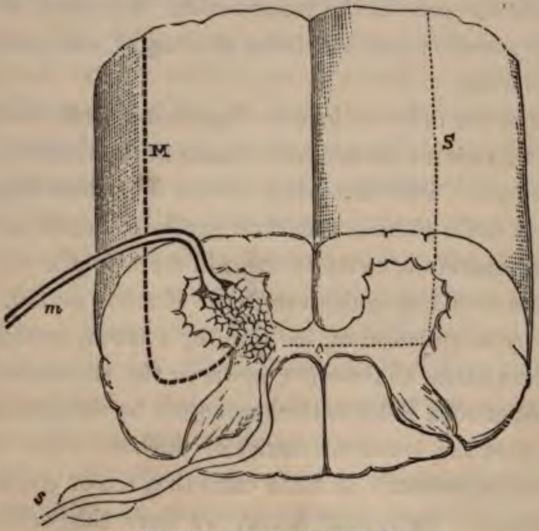

FIG. 24.—DIAGRAM OF A REFLEX LOOP.

M, conducting motor tract; *m*, anterior, motor, nerve-roots; S, conducting sensory tract; *s*, posterior, sensory, nerve-roots.

similar wide diffusion may occur in pathological states of the cord. These reflex loops are also connected with the conducting tracts to and from the brain. A motor impulse, passing down the cord in the white column (M), leaves the cord by the anterior roots (*m*), which are part of the reflex loop, and enters the anterior roots by the gray matter and motor nerve-cells, which may be regarded as part of the reflex centre. So, too, the sensory impulse enters the cord by the posterior nerve-roots (*s*), which are also part of the reflex loop, and then, leaving this loop, ascends the opposite side of the cord to the brain. Thus, the same peripheral impression excites a conscious sensation and a reflex action; and, on the other hand, we can, if we wish, execute voluntarily a movement of the leg quite the same as the reflex action. Moreover, we can exercise some voluntary control over the reflex movement, and prevent or lessen the start of the leg.

The value of the reflex actions in diagnosis is, that their persistence is proof that there is no considerable disease in the reflex loops by which they are produced. They thus yield us very important diagnostic information. Both their absence and their excessive degree are significant. It will be well, therefore, to study them in more detail.

It is necessary to distinguish two forms of reflex action. The first is that excited by stimulation of the skin, by a touch, scratch, prick, etc. On gentle stimulation, contraction occurs in the muscles at or near the spot. A series of such reflex actions can often be obtained in the normal spinal cord, from the lowest extremity of the cord to the lower part of the cervical enlargement. In some cases they are of considerable diagnostic importance. Beginning below, we have the well-known reflex from the sole (plantar reflex) which depends on the lower part of the lumbar enlargement, when the movement which results is confined to the foot-muscles. (See Table, p. 257.) Next, irritation of the skin of the buttock, in some individuals, excites a contraction of the glutei—the gluteal reflex, we may call it—depending, I believe, on the cord at the level of the 4th or 5th lumbar nerve. Next, there is the well-known cremaster reflex, by which the testicle is drawn up when the skin on the inner side of the thigh is stimulated. This arises at the level of the 1st and 2d lumbar pairs. It may often be excited by stimulation of any part of the front and inner side of the thigh.[1] Next, there is the abdominal reflex—a contraction in the abdominal muscles, when the skin is stroked on the side of the abdomen, from the edge of the ribs downwards. This is produced in the cord from the 8th to the 12th dorsal nerves. Next, a stimulation on the side of the chest, in the 6th, 5th, and sometimes in the 4th intercostal spaces, causes a dimpling of the epigastrium on the side stimulated. I think that it depends on a contraction in the highest fibres of the rectus abdominis; it is singularly uniform in its occurrence. We may term it the epigastric reflex; it depends on the spinal cord from the 4th to the 6th or 7th pairs of dorsal nerves. There is no higher reflex on the front of the trunk. If we turn to the back, we shall find that in some patients, from the angle of the scapula to the iliac crest, stimulation of the skin along the edge of the erectors of the spine excites a local contraction in these muscles. These dorsal and lumbar reflexes, as they may be termed, are only of corroborative value, as they are less active than the more convenient abdominal and epigastric reflexes, which are produced in the same region of the cord. Irritation of the skin in the interscapular region gives us, however, the highest reflex available—a contraction in some of the scapular muscles, when slight, chiefly marked at the posterior axillary fold (teres); when more considerable, involving almost all the muscles attached to the scapula—trapezius, teres, serratus—and even moving the bone a little outwards. We may term it, therefore, the scapular reflex, and it is produced in the cord at the level of the upper two or three dorsal and lower two or three cervical nerves.

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[1] This reflex has been carefully studied by Jastrowitz, and by Weir Mitchell.

Thus in these reflexes—plantar, gluteal, cremasteric, abdominal, epigastric, and scapular—we have the means of ascertaining something of the condition of almost every inch of the spinal cord from the cervical enlargement downwards. The presence of the reflexes is proof that the reflex path through the cord is not seriously uninterrupted, but we cannot *simply* infer from their absence that this path is impaired. The reflex excitability of the cord varies much in different individuals, is always greatest in early life, and is often lessened in the old. Some of these reflexes are thus absent, apart from disease, especially the gluteal and lumbar reflexes, and sometimes the cremaster reflex, the abdominal reflex is also lessened by laxity or distention of the abdominal parietes. It is a remarkable fact, also, that disease of one cerebral hemisphere lessens or abolishes these superficial reflexes on the opposite (paralyzed) side of the body. The diminution may be observed immediately after the occurrence of the cerebral lesion, and may be permanent. It is an effect very difficult to explain, because these reflexes are increased if the disease, which lessens voluntary power, is not situated in the brain, but is high up in the cord.[1] The effect of cerebral disease does not interfere materially with the use of these reflexes as indications of spinal disease, and it affords us an important additional indication of the existence of an organic disease of the brain. I will presently give you some instances of the utility of these reflexes in spinal diagnosis.

The second group of phenomena which depend on reflex action are those which have been termed "tendon-reflexes." These phenomena are of great practical importance, and it is necessary to describe their character and nature in some detail.[2]

We will first consider the well-known jerk of the leg which occurs when the patellar tendon is tapped. It has been called the "knee phenomenon" by Westphal, the "patellar-tendon reflex" by Erb. The latter designation has come into general use, although, as we shall presently see, it is an undesirable term. We may, therefore, speak of it either by the somewhat cumbersome designation proposed by Westphal, or by the simpler descriptive term "knee-jerk." It is not a little curious that this knee-jerk, which has for generations amused schoolboys, should have become an important clinical symptom.

To obtain the jerk, the knee must be flexed so that the quadriceps femoris is gently extended, and the leg must be free to move. If then the patellar tendon is struck, the quadriceps contracts and jerks the leg forwards. The most convenient position is with the knee to be tested flexed nearly, but not quite, at a right angle. The posture commonly employed is with the leg to be tested across the other, the knee of the supporting leg being at a right angle (Fig. 25). But if the leg to be tested is stout, its tension in this position may be too great to permit of any movement. In such case the best posture is for the observer to place his arm beneath the patient's thigh, just above the knee, and rest his hand on the patient's other knee (Fig. 26). Not long ago I saw a rather stout man, well known to many members of our profession, who was uneasy because a physiological friend had been unable to produce this phenomenon upon him. His legs were so stout that, in the posture commonly employed, no movement occurred when the patellar tendon was struck. But when the thigh rested on the observer's arm, in the way I have described, the tap on the tendon caused a ready jerk, much to the satisfaction of the individual examined, whose anticipations of impending locomotor ataxy were thus removed. Children may sit on the edge of a chair, adults on the edge of a table; but if so, and the legs are vertical, the effect of the blow and of the muscular contraction must be carefully distinguished. The side of the extended hand is a convenient instrument for giving the blow (Fig. 25). Now and then, when very slight, a percussion hammer (Fig. 26), or a stethoscope with an india-rubber edge to the ear-piece,[1] elicits it more readily, especially when (as in children) the space between the patella and tibia is too small to permit of a suitable blow with the hand. It may commonly be obtained as readily through one or two garments as upon the skin. If its existence is doubtful, however, the skin should be bared. In many cases the movement may be obtained by a downward blow upon the patella, by a blow on the quadriceps tendon above the patella, or by a blow on the substance of the muscle, almost as readily and strongly as by a blow on the patellar tendon. In cases in which it is in great pathological excess, it may even be excited by a blow on the tibia.

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[1] There is, I think, only one possible explanation. In the frog the superficial reflexes are controlled by a centre, situated in the optic lobes, and are lessened, or at least retarded, if this is stimulated. It is probable, as just observed, that there is also in the higher animals a centre which has the power of controlling these reflex actions. If we assume that this controlling centre is itself under the influence of the highest motor centres—not an improbable assumption—all the phenomena are intelligible. The motor centres nominally restrain the controlling centre: if the motor centres or path from them to the controlling centre) are damaged, this centre is unrestrained, and inhibits the superficial reflexes on the paralyzed side. But disease in the cord interrupts, not only the voluntary path, but also that by which the controlling centre influences the superficial reflexes and so these are intensified in the paralyzed parts.

[2] First systematically studied by Erb and Westphal, but previously partially recognized and employed in diagnosis by Charcot.

When it is in excess, the same phenomenon may be conveniently brought out in a somewhat different way. As the patient lies in bed, the finger of one hand is placed across the quadriceps tendon just *above* the patella, and the patella pushed down, so as to make the quadriceps tense. The finger is then percussed in the direction in which the patella is being pushed, so as, suddenly, to increase the tension in the muscle. The blow is instantly followed by a contraction, jerking the patella and finger upwards. Very often this single contraction is immediately succeeded by a second, and this by a third, and so on—a series of quick clonic contractions, recurring as frequently as eight per second. By grasping the patella firmly, and suddenly pushing it downwards, so as to make the muscle tense, this clonus may also be set up, as Erb has shown. It may continue as long as tension is kept up, but instantly ceases when the muscle is relaxed.

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[1] Such stethoscopes were formerly in use for percussion of the chest, and can be obtained of most instrument makers.

The next important phenomenon belonging to this group occurs at the ankle-joint. If the calf-muscles, which are connected with the Achilles tendon, are made tense, and this tendon is tapped, the muscles contract, causing a slight extension movement of the foot; just as the muscles of the thigh contract, when the patellar tendon is struck. In cases in which these phenomena are excessive,—just as sudden tension in the thigh-muscles will cause a contraction, followed by others in a continuous series—so, in such cases, if the calf-muscles, which extend the ankle-joint, are suddenly put on the stretch by pressing the hand against the sole of the foot (Fig. 27), a quick contraction occurs, instantly ceasing, but, if the pressure is kept up, instantly renewed, and recurring, as long as the tension is maintained, as a clonic series of spasmodic contractions—the "ankle-clonus," or "foot-clonus" (or "foot phenomenon"—Westphal). It can often be obtained best when the knee is not

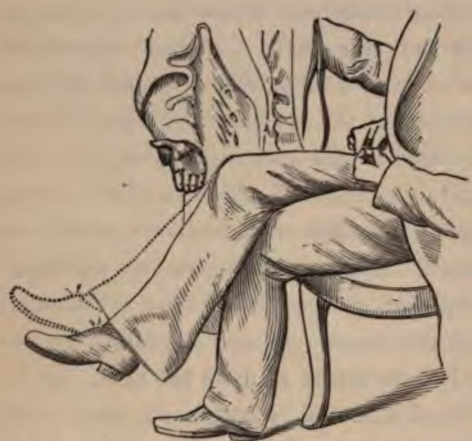

FIG. 25.—ORDINARY MODE OF OBTAINING THE THE KNEE-REFLEX.

The dotted line indicates the movement which follows the blow on the patellar tendon.

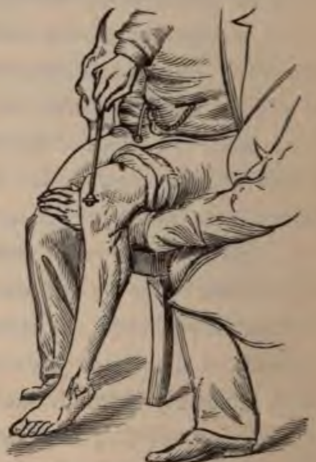

FIG. 26.—KNEE-REFLEX.

Method of obtaining it with a percussion hammer when it is not readily produced in the ordinary way.

completely extended. The movement is very uniform, from six ten contractions occurring per second. By attaching a writing point to the foot, and making it trace a line on a revolving cylinder covered with blackened paper, I have obtained such tracings as I now show you (Fig. 28), which are almost as regular as the tracings of a tuning fork. This foot-clonus can be more frequently obtained than the clonus in the extensors of the knee, but the two have the same time, and are evidently of the same nature.

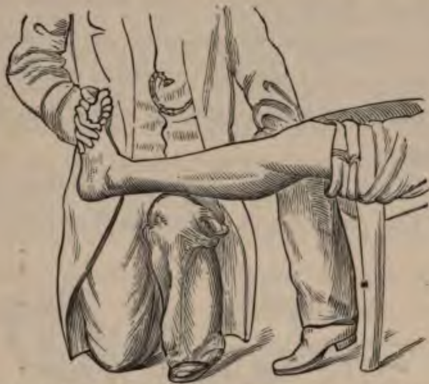

FIG. 27.—METHOD OF ELICITING THE FOOT-CLONUS BY PASSIVE FLEXION OF THE FOOT.

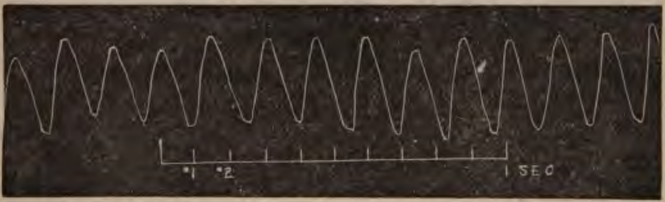

FIG. 28.—TRACING OF THE FOOT-CLONUS IN PARAPLEGIA.

(The tracing reads from right to left.)

What is the nature of these phenomena? When a tendon is tapped, and its muscle contracts, the occurrence has somewhat the aspect of a reflex action. It was assumed by Erb that the contraction is a true reflex action, the stimulus being the excitation of nerves in the tendon. Hence it has been termed "tendon-reflex." This view has received apparent confirmation by the discovery of certain facts: (1) That there are nerves in tendon.[1] (2) That these phenomena depend for their occurrence on the integrity of the reflex path to, through, and from the spinal cord, and are arrested by a lesion in this path. By experiments on animals (in whom similar contractions may be obtained) it has been found that they are prevented by division of the nerves to the muscles, by division of either the anterior or posterior roots of the spinal nerves, or by destruction of the spinal cord.[1] The knee-jerk cannot be obtained in locomotor ataxy (damage to the posterior nerve-roots), or in infantile paralysis (damage to the gray matter, the reflex centre). (3) That these phenomena are in excess in some cases, in which the reflex action from the skin is in excess.

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[1] It does not need the microscope to demonstrate this, as any one may ascertain who will take the trouble to give his Achilles tendon a sharp pinch.

These facts certainly prove that some reflex influence is concerned in the production of the phenomena. But (as Westphal has always maintained) they do not necessarily prove that the contractions depend on a simple reflex action from the tendons. A little consideration of the facts which I have already described, will show that there is a good deal which cannot be explained by the "tendon-reflex" theory. There is the great fact that passive tension is necessary for the tap on the tendon to be effective, and that when the phenomena are in excess, sudden tension alone will suffice to develop the contraction. But tension acts upon the muscle as well as upon the tendon, and frequently the contraction may be distinctly excited by stimuli which act on the muscle and have no action on the tendon. For instance, in cases in which these phenomena are in excess, if the foot be gently pressed up so as to make the calf-muscles tense, and the muscles on the front of the leg be

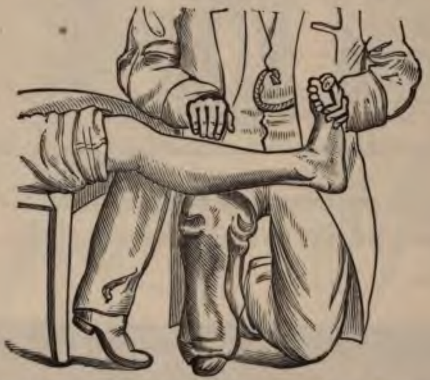

FIG. 29.—METHOD OF OBTAINING THE FRONT-TAP CONTRACTION.

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[1] See Tschirjew: "Archiv für Psychiatrie," Bd. VIII., Heft 3.

tapped, the calf muscles contract, just as they do when, under the same circumstances, the tendon is tapped, and cause a brief extension movement of the foot. I have termed this the "front-tap contraction." It is a very delicate test of increased irritability, and it is also of considerable theoretical interest, since we have in it a contraction developed by a stimulus which does not in any way affect the tendon. It can affect the gastrocnemius directly, for, by placing the hand on the calf, a vibration may be felt through the leg. If the tibia, instead of the muscle, is tapped, the contraction is much slighter, or does not occur.

Moreover, a tap on the tendon itself only excites the contraction when it increases the tension of the tendon; *i.e.*, when it acts upon the muscle also. In the case of the Achilles tendon, this may be easily demonstrated with a little care. A gentle tap on the side of the tendon will excite the contraction as readily as a tap on the back of the tendon, but if the other edge of the tendon is supported (as by the fingers of an assistant), the same tap will no longer be effective, because it no longer increases the tension.

The strongest proof, however, of the independence of the phenomena on any stimulation of the tendon is afforded by the experiments of Tschirjew, who divided carefully all the nerves to the patellar tendon, and still found that the tap upon it made the tense muscle contract.

Thus the evidence seems conclusive that the contractions are not excited by stimulation of the nerves of the tendon, but that the stimulus originates in the muscle, the tendon being only, so to speak, an instrument by which that stimulation is produced.

But if the muscle is stimulated and then contracts, is not the contraction excited locally, as Westphal has, from the first, urged? A reflex action takes a certain time, which is needed for the stimulus to travel to and from the cord, and for the reflex process to occur in the centre. According to received physiological data, an interval of at least one-fifteenth of a second would be needed for the knee-jerk, if it were a reflex process, and rather more for the movement at the ankle. I have found that when the Achilles tendon, or the front of the leg is tapped, the resulting contraction occurs in about one-thirtieth of a second.[1] The interval for the knee-jerk has been found to be about one-twenty-fifth or one-thirtieth of a second.[1] If the patella is pressed down and tapped, in the way I have just described, I have found that the interval between the tap and the resulting contraction is often not more than one-fortieth of a second. The shortness of the interval makes it difficult to believe that these contractions can be reflex, and supports the theory that they are excited locally.

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[1] "Med.-Chir. Trans." 1879, p. 292. The measurement has been since confirmed by Waller, "Brain," July, 1880.

But to this view, that the contractions are excited locally, is apparently opposed the fact that they are prevented by whatever lesion arrests reflex action. Some have endeavored to explain the discrepancy by the suggestion that reflex action may occur in a much shorter time than is commonly supposed. This suggestion is not at present justified by any known facts. Another and, I think, much more probable explanation is this. If we regard the contractions as local, we have still to account for the irritability which permits the local stimulus to cause a contraction. This irritability is developed by passive tension. If the muscle is relaxed the fibres may contract if they are struck directly, just as do the fibres of a separated frog's muscle, but no contraction can be produced by striking the tendon. Hence I have suggested that the tension excites, by a reflex influence, a state of extreme irritability to local stimulation—such as that of a tap on the tendon, or such as the vibration from a tap near the muscle, or from a tap on the bone to which the tendon is attached—which thus excites a visible contraction.[2]

The explanation receives some confirmation from the very interesting observations of Tschirjew.[3] He has found that if the nerve to a separate muscle be divided, the muscle remains of just the same length. If, however, a weight be first attached to the muscle, when the nerve is divided the muscle lengthens. This shows that the tension does excite a slight contraction which is dependent on a central influence. It is in this condition only that the local stimulation is effective. If the tension put on a muscle is gentle and gradual, it may only develop the irritability, and an additional local stimulation is necessary to produce a visible contraction. If, however, the tension is sudden and forcible, it not only develops the irritability, but produces visible contraction in the muscle thus rendered irritable—as in setting up the foot-clonus. I have shown[1] that the relaxation of the muscle, between the successive contractions, is not complete: there is a persistent residual contraction, *i.e.*, a tonic contraction on which the clonic contractions occur. When one clonic contraction is over, the tension continuing, a second is instantly developed.

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[1] .039 sec. Burkhardt; .032—034 sec. Tschirjew; .04 sec. Brissaud; .03—.04 sec. Waller; .3 sec. Eulenberg. Some measurements which I made of the interval ("Med.-Chir. Trans." 1879, p. 275) gave a longer interval, probably in consequence of the movement of the foot being taken as the indication of the commencing contraction. "Load" will increase greatly the period of latent stimulation, probably by causing the initial contraction to expend itself on the elasticity of the muscle. The measurements given above were obtained by recording the commencing contraction of the muscle.

[2] This explanation was originally given for the "foot phenomenon" alone ("Med.-Chir. Trans." 1879, p. 295). I now think that it is equally applicable to the knee-jerk. The evidence of the identity in nature of the two is very forcibly stated by Waller (loc. cit.).

[3] "Reichert und Du Bois Reymond's Archiv," 1879.

The sensory nerves of muscles have been shown by Tschirjew to commence, not within the muscular fibrils, but in the interstitial connective tissue. The afferent impulse produced by tension is apparently due to the tension acting on these nerves: the visible contraction is excited by tension or vibration affecting the muscular fibres themselves. The latter is ineffective unless the muscles are brought into a state of special excitability through the cord. Of the reality of an afferent impulse from the muscle, produced by tension, you may easily convince yourselves, if you will allow your ankle to be suddenly flexed. A distinct pain is felt in the muscle (none, be it observed, in the tendon). It is not surprising, therefore, that this afferent impulse should, very often, not merely develop the reflex excitability, or tonic contraction, but also cause a more widely-spread reflex action. The attempt to get the foot-clonus, for instance, will cause a flexion of the hip-joint; the attempt to obtain the knee-jerk may cause a movement in the opposite leg or a start back of the body. But these reflex contractions, if carefully observed, confirm the theory which has been put forward in the preceding pages, for they distinctly succeed, at an appreciable interval, the local contraction. If, for instance, in a patient now under my care, I depress the patella so as to make tense the quadriceps, and then tap the depressing finger, the tap is followed, after an interval too short to be recognized, by a contraction in the muscle, and after a very distinct interval (which I have found to be about three times as long as the other), by a contraction in the opposite leg. So, too, Burckhardt has found that the latent interval for a skin-reflex is three times as great as for the knee-jerk. I think that this theory of reflex irritability and local stimulation affords a full explanation of all the relations of these phenomena to the central nervous system, and to the other phenomena of disease, and it is the only theory which adequately explains them.

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[1] Loc. cit. p. 286.

It seems, therefore, most desirable to discard the term "tendon-reflex" altogether. The phenomena are, according to the explanation above given, dependent on a "muscle reflex" irritability, which has nothing to do with the tendons. If we wish to describe them by a general term, it will be best to employ one which does not involve any special theory of their nature. They may be termed "tendon-muscular phenomena," but the intervention of tendons is not necessary for their production; the one condition which all have in common is that passive tension is essential for their occurrence, and they may more conveniently be termed *myotatic* contractions (τατικος, extended).[1] The irritability, on which they depend, is due to and demonstrative of a muscle reflex action which depends on the spinal cord.

A true "tendon-reflex" may be excited by pinching the tendon, but this is a start of the whole limb, precisely such as results from a pinch of the skin.

A clonus quite similar to that just described can be sometimes obtained in the peronei (a lateral foot-clonus), and also in the plantar muscles of the great toe—in each case by passive tension. All have nearly the same time—about eight per second.

It is of interest to note that modern physiologists know nothing of muscular "tone" except as developed by tension, and it is highly probable that the condition on which the myotatic irritability depends, is identical with muscular "tone." It is not, therefore, surprising that we are able, even in health, to get evidence of a tendency to rhythmical contraction. If a rhythmical contraction can be set up voluntarily, and gentle tension in the gastrocnemius maintained, as by sitting on the edge of a chair with the ball of the foot resting on the ground, the contractions will go on involuntarily—a normal foot-clonus, which has precisely the same time (about six per second) as the morbid clonus (Fig. 30). A prolonged voluntary contraction of the calf-muscles against a considerable resistance (as in standing for some time on tip-toe) is also broken into clonus. These are evidently the same phenomenon, the difference being that it cannot in health be excited by passive tension. For *this* to be effective a morbid reflex irritability is needful, such as only exists in disease. In morbid states, in which the myotatic irritability is excessive, the posture I have just described excites the clonus very readily; and the jerking legs of paraplegics as they sit must be familiar to you. In attempting to walk, also, the tension on the calf-muscles has the same effect, and the patient may be jerked violently by the spasm.

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[1] If it should ultimately be proved (which is very improbable) that so short an interval as one-fortieth of a second is sufficient for a reflex action, and that each contraction is reflex, the term "myotatic" will still be accurate, since it will remain true that tension is essential for the production of these contractions.

It is probable that this reflex relation between tension and contractility of muscles is of the highest importance in their associated action, and that the reason why, in certain muscles, as those of the calf, these phenomena are more readily observed, is because in these, during the process of walking, at every step contraction succeeds tension, and so the reflex relation between the two has attained a higher degree of development. It is rare to get a distinct clonus in a child who has never walked, even in the pathological conditions in which in adults it would certainly be obtained.

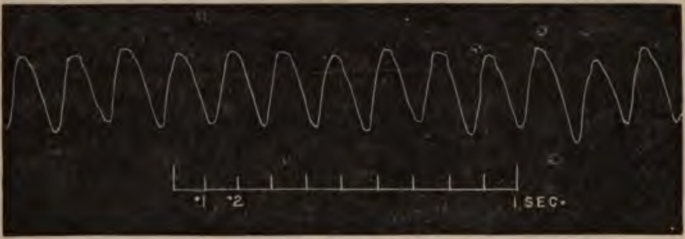

FIG. 30.—NORMAL FOOT-CLONUS.
(The tracing reads from right to left.)

The excess of these myotatic contractions is especially related to degeneration in the lateral column of the cord (pyramidal tracts). It is seen in extreme degree, for instance, in lesions of the cord higher up, which cause descending degeneration in those columns. It is seen also in cases of hemiplegia with similar descending degeneration on the paralyzed side. The excess is apparently not due to the simple loss of a cerebral influence. It does not, as a rule, occur immediately, but develops gradually at the end of a week or ten days, *i.e.*, after a time sufficient to allow the nutrition-changes of commencing degeneration to pass down the lateral (pyramidal) fibres to the neighborhood of the muscle-reflex centres. Over-action of these centres follows, and must be ascribed to the loss of some controlling influence. But the control cannot be exerted by the fibres themselves, or the over-action due directly to their degeneration, since the fibres only conduct, and do not originate nerve force. The pyramidal fibres enter the gray substance, and apparently end in its interlacing network of fine fibres and minute nerve cells, and by means of this structure are connected with the large ganglion cells of the motor nerves. The phenomena are best explained by the hypothesis that this structure, in the matrix of the gray matter, controls the muscle-reflex centres (of which the ganglion cells are part), and that the degeneration of the pyramidal fibres, which can be traced to the gray matter, invades this structure. The loss of its influence permits the over-action of the muscle-reflex centre. The foot-clonus can often be obtained in slight chloroform narcosis, but under the full action of chloroform it disappears; apparently this agent influences the controlling structure before it influences the muscle-reflex centres. Ether and nitrous oxide do not abolish myotatic irritability (Horsley). After an epileptic fit, again, a clonus can be obtained for a few minutes, while after a very severe fit, the myotatic irritability, instead of being increased, may for a few seconds be lost, so that not even the knee-jerk can be obtained. In the first case the discharge appears to temporarily exhaust the controlling structure; in the second, the more severe action exhausts also the muscle-reflex centre itself.[1]

It is uncertain whether we should ascribe all over-action of these centres to loss of control. We are justified in doing so when the over-action follows permanently a destroying lesion, such as the degeneration of the lateral columns. Slight over-action is sometimes present when there is no evidence of such a process, but in which there are indications of irritability of the spinal cord, as in cases of hysteria with spinal tenderness. It is possible that in these cases we have a primary over-action of irritable centres.

The diagnostic importance of a considerable increase in myotatic irritability can hardly be over-estimated. By "considerable increase" I mean such a degree that a uniform clonus can be obtained by simple passive flexion of the foot. I believe that such a clonus is always pathological, always indicates grave nutritive changes in the spinal cord, and that there is, in most cases in which it can be obtained, actual degeneration in the fibres of the lateral columns.

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[1] I do not know of any pathological observations of the state of the matrix of the gray substance when there is degeneration of the lateral columns without affection of the motor ganglion cells. The investigation is very difficult on account of the character of the structure. In cases so extremely rare as to be manifestly exceptional in mechanism, the foot-clonus has been observed a few hours after the onset of hemiplegia. It is probable that in these cases the controlling structure is inhibited by the irritation of the cerebral lesion. A case lately seen suggests that the knee-jerk may even be lost during the first hour after the onset, and then return: the irritative inhibition extending to the muscle-reflex centre itself.

A circumstance which gives to this sign a special diagnostic importance, is that in the cases in which it occurs the nutrition of the muscles and sensibility of the skin are often unimpaired, and the weakness in the legs is likely to be regarded as "functional," or, if in a woman, as "hysterical." In hysterical paraplegia the myotatic irritability is often perfectly normal. It is not uncommon, however, in these cases, to have, with spinal tenderness, an increased knee-jerk, but I have never seen, in any case of hysterical paraplegia, a foot-clonus such as is above described, and is so common in organic cases. But a spurious, or voluntary foot-clonus is occasionally met with in hysteria, which needs to be carefully distinguished from the typical form. In the latter, when the foot is first pressed up, the clonus at once commences and continues without variation as long as the pressure is maintained. But in the voluntary form there is no clonus during the first few seconds, then the foot and observer's hand are pressed down by a voluntary contraction in the calf-muscles which is broken by clonus. This clonus may continue, but varies in degree (together with the pressure downwards of the foot) from time to time. This variation and the dependence of the clonus on voluntary contraction of the calf-muscles, can be readily recognized.

Similar myotatic contractions may be obtained in the arm. A tap on the extensor tendon above the olecranon will cause a contraction in the triceps. When they are in excess a tap on the bone to which the the tendon is attached will cause a contraction in the muscle. Thus a tap on the radius will cause a contraction in the biceps, slightly flexing the elbow; a tap on the ulna will produce contraction in the triceps, or in the forearm muscles connected with the bone, just as a tap on the tibia, in similar conditions, will cause the knee-jerk. A clonus may even sometimes be obtained in the biceps, or flexors of the fingers, by sudden tension, quite similar in time and character to the ankle-clonus. In many cases these contractions can be obtained without any other passive tension than is involved in the posture of the arm. In hemiplegia the increased muscular tension which is involved in the condition of rigidity seems to suffice for permitting their occurrence. Each contraction is excited by the mechanical stimulation of the jar or of the increase of tension produced by the tap.

The myotatic irritability is lost in diseases which separate the muscle from the spinal cord (as a lesion of the nerve), in disease of the posterior roots (such as exists in posterior sclerosis), in disease of the anterior roots (as in chronic meningitis compressing the nerve-roots), and in disease of the gray matter at the level from which the nerves for the muscle proceed (as in infantile paralysis and allied diseases). It is also lost in diphtheritic paralysis[1] (in which there is an affection of the motor-nerves and their cells) and in pseudo-hypertrophic paralysis (probably by reason of the disease in the interstitial tissue of the muscle in which the afferent nerves begin).

In employing these contractions for purposes of diagnosis, it must be remembered that they are, in health, similar on the two sides. A difference between the two sides is always pathological. For instance, the front-tap contraction can sometimes be obtained in persons in whom there is no reason to suspect organic disease. But the presence of the front-tap contraction on one side, and its absence on the other, shows, without doubt, some structural change. Not long ago I saw a young married lady suffering from weakness of the legs. She was very anæmic, and the paraplegia had been regarded as unquestionably "hysterical," and she had been urged to exert herself and shake it off. The only absolute objective sign was that the front-tap contraction was very well marked on one side, and entirely absent on the other. This was strong evidence of structural change in the cord. This diagnosis was confirmed, only too sadly, by the subsequent progress of the case, for I afterwards heard that the patient became completely paraplegic, bed sores formed, and it seemed likely that she would die.

Before leaving these spinal reflex phenomena, I may, parenthetically, illustrate, by an example, the manner in which they are changed in cerebral disease, and the occasional diagnostic importance of their alteration. The cutaneous reflexes, especially the trunk reflexes, are often lessened, but the myotatic contractions are increased when there is any degree of descending degeneration in the cord. Under these circumstances the foot-clonus and front-tap contraction may be obtained. Not long ago, in investigating these reflexes, I examined a man who was thought to be suffering from idiopathic epilepsy. No weakness was complained of. The epigastric and cremasteric reflexes were, however, absent on the right side, and the abdominal reflex was slight, although all were well marked on the left. In the right leg the knee-jerk was excessive, and the foot-clonus and front-tap contraction could readily be obtained, while they could not on the left. This alteration affords the strongest grounds for suspecting organic brain disease, and its discovery led to careful examination of the strength of the right limbs. Slight but distinct weakness of the right arm and leg was found, and subsequently increased, and optic neuritis developed. In this case organic disease of the brain might readily have been overlooked, and probably would have been overlooked for a time, had it not been for the indications afforded by these phenomena.[1]

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[1] Erb, Buzzard. I have several times observed the same fact.

Co-ordination of Movement.—The next function of the spinal cord to be mentioned is that by which it influences the co-ordination of muscular movements. This function apparently depends upon the posterior columns, for it is lost when there is disease in this situation, as in locomotor ataxy. It is not, however, the whole of the posterior columns which are related to the function, but merely the part distinguished as the postero-external column or root-zone, that through which the fibres of the posterior roots pass. The most distinct ataxy may result from disease limited to this situation (Plate, Fig. 5).

Why does disease of the posterior columns interfere with co-ordination? We have seen that, by means of the muscular reflex actions, muscular contractions become associated; tension influences contractility; and thus there is a reflex grouping of muscular actions, which undoubtedly plays a very important part, not only in the actual arrangement of the contractions, but also by, so to speak, moulding the spinal centres by establishing lines of lessened resistance through them, and so facilitating the voluntary co-ordination.[2] There is, I believe, another mechanism by which the impairment of reflex action may impair co-ordination. For any movement there must be not only a contraction of certain muscles, but also a proportioned relaxation of their opponents. There is reason to think that the relaxation is really due to a reflex action,[1] and this is also suggested by the fact, long ago pointed out by Duchenne, that the late rigidity of hemiplegia may commonly be readily relaxed by faradization of the opponents of the rigid muscles. If this is so, we obtain a glimpse of a very complex series of reflex actions, by which a relation is established between tension, contraction in the same muscles, and relaxation in their opponents, which must undoubtedly play a very important part in muscular co-ordination. These muscle-reflex processes in locomotor ataxy are almost always impaired (if we are to judge by the knee-jerk, which is commonly and early lost). Their loss is probably one factor in the inco-ordination of ataxy. It is possible also that, as Todd long ago taught, these postero-external columns contain fibres which connect groups of nerve-cells at different levels, and that upon this connection depends in part the association of muscular contractions, which is essential for co-ordinated movement. The impairment of this association, by posterior sclerosis, may be another element in the ataxy.

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[1] In this patient the right hemiplegia and the optic neuritis increased, and ultimately left hemiplegia gradually supervened. After death there were found tumors in each hemisphere of the brain. That on the left side, which was the larger, the size of a pullet's egg, was situated above the lateral ventricle, beneath the upper extremity of the ascending frontal convolution, and it had extended through the lateral ventricle to the surface of the optic thalamus. It had clearly interrupted the connection of the upper "motor convolutions" with the motor tract.

[2] The springing movements observed by Woroschiloff in the hind legs of the

But there are cases in which ataxy exists without loss of the knee-jerk, and with the foot-clonus. In these cases there is always loss of power as well as inco-ordination, and there is sclerosis of the lateral columns as well as of the posterior. The lateral sclerosis will tend to increase the muscle-reflex action, and, apparently, the damage to the posterior roots is then insufficient to arrest it, or the sclerosis is so situated as not to affect the posterior roots, although it may perhaps interfere with the connection between different groups of nerve-cells. It may be remarked that the inco-ordination is in these cases never quite like that in typical ataxy—there is more unsteadiness than inco-ordination. In other symptoms also these cases differ from those of pure ataxy. "Lightning pains" are rare and sensation is usually normal.

When superficial sensation is lost from the extensive damage to the posterior roots, this loss will greatly increase the ataxy by the removal

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rabbit after section of the dorsal cord, and regarded by him as "co-ordinated" in the lumbar enlargement, were probably merely a consecutive series of muscle-reflex actions, analogous to the slow knee-clonus which I have described ("Med.-Chir. Trans." 1879, p. 289).

[1] See "The Movements of the Eyelids."—"Med.-Chir. Trans." 1879.

of an important indication for cerebral guidance of movement. But since ataxy may exist without loss of superficial sensation, this is evidently not the *chief* element in the condition.

In the inco-ordination of movement which depends on the spinal cord, we greatly intensify the manifestation of the trouble by reducing the base of support, since the smaller this is, the more accurate is the adjustment of muscular actions that is needed to maintain perfect equilibrium. Hence the ataxic has a difficulty in standing with his toes and heels close together; and if his feet are uncovered, the irregular muscular contractions are shown by the twitching of the tendons. Moreover, we increase his difficulty by making him close his eyes,—withdrawing, thus, the visual guidance. It has been said that this test is only effective when sensation is impaired in the legs, *i.e.*, when the sensory impressions from the legs are insufficient for cerebral guidance. This is, however, not true. The effect may be very marked when sensation is perfect. The maintenance of equilibrium is partly a muscle-reflex act, and if this function is slightly impaired, we can easily understand that it should be inadequate, unless supplemented by the visual information. Even when there is no inco-ordination, under perfectly normal conditions, closure of the eyes causes slight unsteadiness. In ataxy this is, so to speak, multiplied by whatever defect may exist.

It is important to be aware that the inco-ordination may not be equally distributed through the legs. In some patients it is marked in the muscles of the hips and knees, and the legs are raised too high, and brought down too suddenly. In other cases this characteristic is wanting; the inco-ordination affects chiefly the muscles of the feet, and causes unsteadiness of gait and irregular action of the foot-muscles in standing, or in movement of the feet, very conspicuous when they are uncovered.

Ataxy may be manifested, not only in the muscular contractions which produce movement, but also in those which should keep the limbs in a fixed position. It may be conspicuous, as already stated, in the attempt to stand upright. If the arms are affected it may be seen when the patient holds his hands out and tries to keep them in the same posture. Slow involuntary movements of the hands and fingers take place. These are increased in degree by closure of the eyes, and may resemble the movements of athetosis. The patient is unaware of their occurrence.

Controlling Functions.—We may consider next the controlling functions of the cord; and first, the influence which it exerts over nutrition. The nutrition of the limbs, etc., is, to a considerable extent, under the influence of the cord; that of the muscles, and probably also of the bones and joints, through the anterior nerve-roots; that of the skin probably through the posterior.

Muscular Nutrition.—For diagnostic purposes the most important is the influence on the nutrition of the muscles. The path of the influence is the motor-fibres in the anterior roots and nerve-trunks. Changes in the nutrition of the muscles, which are not due to local influence, depend on changes in the nutrition of the motor nerve-fibres. But the motor fibres are the prolonged processes of the motor nerve-cells, and may be regarded as parts of the nerve-cells, sharing all changes in the nutrition of the cells. The nerve-fibres are excitable by electricity, and changes in their nutrition are accompanied by changes in their excitability. By the use of electricity we are thus able to ascertain their state of nutrition, and to learn what is the condition of the nerve-cells in the cord, provided there is no disease separating the part of the fibre tested from the influence of the cells. Hence the value of electricity in the diagnosis of diseases of the spinal cord.

In a normal state, if you apply either the faradaic or the voltaic current to a motor nerve, there occurs, as you doubtless know, a contraction in the muscles, continuous when the faradaic current is applied, but, if the voltaic current is used, occurring only when the current commences or ceases to pass—*i.e.*, when the circuit is "made or broken." In proportion as the nutrition of the nerve-fibres is impaired, their excitability is lowered; a stronger current of each kind is required to excite them and cause contraction in the muscles they supply. When their nutrition is much impaired—*i.e.*, when the fibres are "degenerated"—no contraction can be obtained even with the strongest currents.

The changes in the excitability of the muscles are less simple, because in them there are two excitable structures—the terminations of the nerves, and the muscular fibres themselves. Of these the nerve-fibres are the more sensitive to faradization, and the faradaic stimulation of a muscle under normal circumstances is by means of these motor nerve-endings. Thus we find that its excitability corresponds in degree to that of the motor nerve supplying it. The muscular fibres themselves are, even in the normal state, less sensitive to faradization than the nerve, apparently because they are incapable of ready response to stimulus so very short in duration as are the shocks of which the faradaic current consists. (The proof of this consists in the fact that under the influence of curara, which removes the excitability of the terminations of the motor nerve, the muscle requires a stronger faradaic current to stimulate it than in the normal state.) But under these circumstances the slowly interrupted voltaic current stimulates the muscle as readily as in the normal state; a contraction occurs when the circuit is completed or broken—distinctly slower than that which occurs when the nerve-fibres are intact, and due to the stimulation of the protoplasm of the muscular fibres themselves. The fact that, under normal circumstances, the contraction which is caused by the voltaic current is as quick as that produced by the faradaic shock, is ground for believing that, in health, the voltaic, as well as the faradaic current, causes the muscle to contract chiefly by exciting the motor nerve-endings. When the motor nerve is degenerated, and will not respond to faradaic or voltaic stimulation, the muscle also loses all its power of response to the former. Apparently the nerve-degeneration is accompanied by changes in the nutrition of the muscular fibre, by which any power of response to faradization, which it possessed in the normal state, is lost. But the response to the voltaic current remains, and becomes quickly more ready than in health, doubtless in consequence of nutritive changes which develop what the older pathologists called, truly enough, "irritable weakness." Moreover, there may commonly be observed a change in the readiness of response to a certain mode of stimulation with voltaism—a "qualitative" change, as it is termed. In health, the first contraction to occur, on gradually increasing the strength of the current, is at the negative pole when the circuit is closed, and a stronger current is required before closure-contraction occurs at the positive pole. But, in the morbid state we are discussing, closure-contraction may occur at the positive pole as readily as at the negative, or even more readily—and contractions, when the circuit is broken, occur far more readily than in the normal state. This condition, then—faradaic irritability lost, voltaic irritability increased and often changed in quality—is termed the "degenerative reaction," because it occurs when the nerve-fibres are degenerated; and if we test *them* we shall find no response to any stimulus, voltaic or faradaic. It occurs when the nerves are separated from their motor nerve-cells, and if no such separation exists it indicates an acute degenerative change in those nerve-cells. It is well seen in acute myelitis of the anterior cornua (as infantile palsy).

But the motor nerve-cells and fibres often undergo changes in nutrition of a much more chronic character. In this condition the irritability of the fibres is lessened gradually and slowly. The irritability of the intramuscular nerve-endings is lessened in the same degree as that of the nerve-trunks, and we have a diminution to both faradization and voltaism. The nutrition of the muscular fibres is slowly, gradually, impaired; and when the nerve-fibres are much affected the muscular fibres are also. There is no stage in which the nerve-fibre irritability is lost, and the muscle-fibre irritability retained; hence there is no condition of lost faradaic and increased voltaic irritability such as characterizes the degenerative reaction just described. Irritability is changed to the one form of stimulus just as to the other. This condition is seen in many cases of progressive muscular atrophy, and also, in slighter degree, when the nerve-cells suffer, not in consequence of disease primary in them, but as a result of degeneration or irritation spreading to them from above. It is seen, for instance, in the wasting which occurs sometimes in hemiplegic limbs.

Between these two forms there are intermediate conditions, especially in cases of subacute disease of anterior cornua. For instance, the nerves may present normal irritability, and the muscle the increased voltaic irritability and changed order of contractions met with in degeneration. Probably, in these cases, some nerve-fibres are degenerated, and lead to increased irritability of some muscular fibres. In both nerve and muscle the character of the reaction is determined by the more irritable structures; hence it is normal in the nerve and altered in the muscle. This is termed the "middle form" of degenerative reaction. It would be more accurate to call it the "mixed form."

The various changes in irritability have been thought to indicate the existence and various affections of separate centres for the nutrition of the nerves and muscles, apart from, though acting through, the motor nerve-cells. Remembering that the nerves and muscles contain fibres which suffer in different degrees, the phenomena at present ascertained may all be explained on the simpler principle stated, without the assumption of these special centres of the existence of which there is, indeed, no evidence.

Frequently the lowered irritability of degeneration in the nerves is preceded by a slight increase of irritability, very transient when the degeneration is acute, of longer duration when the degeneration is of the slower variety just noticed. Thus, in the early wasting of hemiplegia, increased irritability may be found, slowly giving place to diminution. In some morbid states, again, in which the change of nutrition in the cells and fibres is extremely slight, an increase may alone be discovered. I have found such an increase, for instance, in diseases regarded as functional, as paralysis agitans and chorea, and it is an interesting proof of the molecular changes which underlie, or result from, functional maladies.

In employing electricity as a means of diagnosis, at least one of the electrodes should be small, so as to be able to concentrate the current on a single muscle. Great care must be taken to place these electrodes on corresponding points on the two sides. It is convenient to be able to interrupt the current at the battery, so that the effect of the passage of the current may not be obscured by the mechanical effect of the application of the electrode.[1] In Stöhrer's faradaic battery the current can readily be stopped by pressing the hammer with the third finger, while the rod graduating the current is raised or lowered with the thumb and first finger. By moving the hammer slowly with the finger, we may employ the isolated faradaic shock. (The current consists of a rapid succession of shocks.) The isolated shock is often useful, because it is much less painful than the rapid series of shocks, and is especially convenient in the examination of children. A mechanical interrupter is essential in the case of the voltaic battery, in which the stimulation only occurs when the circuit is made and broken; and no battery is suited for use for diagnostic purposes which does not possess such a means of interruption. A galvanometer, to indicate the actual strength of current passing, is also very useful, since the number of cells employed affords little indication of the current which actually passes through the resistance of the skin, which varies much in different parts.

In examining muscles and nerves we avail ourselves, whenever we can, of the opposite side for comparison, and when we cannot, we must, if there is any doubt, compare the results obtained with those yielded by a healthy individual. Two conditions may be tested—first, the lowest strength of stimulus to which the muscle or nerve will respond; and secondly, the relative degree of response to a stronger current. The former is the more important, but has been insisted on too exclusively, for the latter is important also. If a few fibres of a nerve are healthy, and the others are degenerated, contraction may occur with as weak a stimulus as in the healthy nerve; but if the current be made a little stronger, the contraction in the diseased part may remain the same when that on the healthy side is energetic. Both irritability and power, therefore, should be noted, and also the order of reaction to voltaism.

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[1] The interrupting rheophores (containing a mechanism in the handle for making and breaking contact) are less convenient than they appear. In watching for the first flicker of contraction, it is essential that the reophore should be held perfectly still, and the contact cannot be made or broken in the handle without causing a slight movement of the rheophore, which interferes with the detection of the muscular contraction.

The nutrition of bone and joints also probably depends on the anterior gray matter, but the influence is shown chiefly by the effect of disease in retarding the growth of bones. Now and then in posterior sclerosis (locomotor ataxy), a painless joint affection and brittleness of bones develop. When the upper dorsal cord is affected grave thoracic trouble may occur in a similar insidious way. Dyspnœa may lead to an examination of the chest of a patient who makes no complaint, and one pleural cavity may be found full of fluid.

The nutrition of the skin and subcutaneous tissues depends upon nerves which have their course in their posterior, sensory, roots, but whether there are special trophic fibres is unknown, and the centre on which the influence depends is also unknown. It is doubtful whether simple loss of the function of the posterior roots leads directly to lesions of nutrition. These may result indirectly in this condition; the anæsthesia deprives the patient of sensory information when change of posture is required to prevent damage from pressure. Occasionally, however, sloughing and vesication of the skin occur with extreme readiness, on the least local disturbance, and even with none. This is the case when the lesion is irritative in character, especially in destruction of the cord at the level from which the sensory nerves to the part proceed, and sometimes in disease higher up, as in some cases of acute myelitis.

Micturition and Defecation.—The spinal cord possesses centres, situated in the lumbar enlargement, which preside over the action of the bladder and rectum. They are probably complex reflex centres: that for the sphincter ani is the more simple, but the system of action of each may be the same. In the wall of each viscus we have muscular fibres to expel the contents, and at the mouth a sphincter-arrangement to prevent their continuous evacuation. Fæces or air in the rectum, or urine in the bladder, excites the lumbar centre, and causes two effects—

contraction in the wall and relaxation of the sphincter. This process may be, to a considerable extent, controlled by the will, although we are still ignorant of the precise mode in which the voluntary influence is exerted. But if the volitional path in the cord is interrupted above the lumbar centres, the will can no longer control the reflex processes; as soon as fæces irritate the rectum, they are expelled by the reflex mechanism; as soon as a sufficient quantity of urine accumulates in the bladder, a reflex contraction of the detrusor, and relaxation of the sphincter, cause its escape. The affection of the voluntary path for the sphincters is not always proportioned to that for the legs. If the damage to the cord involves also the sensory tract, the patient is unconscious of the action of the bladder or bowel. If the sensory tract is unaffected, the patient is aware of the process, but cannot control it. It is often said that there is permanent relaxation of the sphincters, but this is true only when the lumbar centres are inactive or destroyed. In this condition, evacuation occurs as soon as fæces or urine enter the bowel or rectum. The urine escapes continuously, instead of being expelled at intervals. The condition is less obvious in the case of the rectum, because there is not such continuous entrance of fæces into the

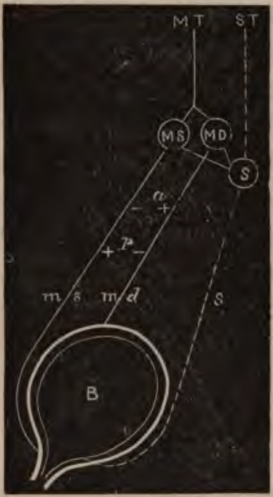

FIG. 31.—DIAGRAM SHOWING PROBABLE PLAN OF THE CENTRE FOR MICTURITION.

MT motor tract, ST sensory tract in the spinal cord; MS centre, and *ms* motor nerve for sphincter; MD centre, and *md* motor nerve for detrusor; *s*, afferent nerve from mucous membrane to S, sensory portion of centre; B, bladder. At *r*, the condition during rest is indicated; the sphincter centre being in action, the detrusor centre not acting. At *a*, the condition during action is indicated: the sphincter centre being inhibited, the detrusor centre acting.

rectum as there is of urine into the bladder. We may, however, distinguish between the two states of the rectum by the introduction of the finger. If the lumbar centre is inactive, there is a momentary contraction, due to local stimulation of the sphincter, and then permanent relaxation. If, however, the reflex centre and motor nerves from it are intact, the introduction of the finger is followed, first by relaxation, and then by gentle, firm, tonic contraction. I have verified this by introducing an India-rubber cylinder instead of the finger, and registering the pressure on the cylinder by connecting it with a recording apparatus, and have found that the relaxation is preceded by a very slight, brief contraction, and is followed by unbroken tonic contraction. The relaxation may also be readily produced by any impression on the mucous membrane of the rectum above the sphincter.

In cases of gradual disease we may often trace the gradual loss of voluntary power over the process of micturition. In some cases this loss of power appears to be manifested as an inability, not to restrain, but to excite the action of the centre, and we have then a tendency to retention. Many conditions can be best understood by assuming that the motor centre really consists of two parts—one (MS, Fig. 31) maintaining the contraction of the sphincter, the other (MD) exciting the contraction of the detrusor fibres, and that these two parts are antagonistic: when one acts, the other is inhibited. Thus, in normal rest, the sphincter centre is active, the detrusor at rest. Action is produced [illegible] a conjoint afferent impulse from the bladder and voluntary impulse from the brain. Then the detrusor centre acts, and the sphincter centre ceases to act. If voluntary power is impaired, the afferent impulse from the bladder may be insufficient, and then there is retention, or in other such cases the motor centre may yield too readily to the afferent impulse, and there is reflex incontinence.

Sexual Functions.—The conditions of the sexual organs depend on the integrity of the reflex loop to and from a special centre, also situated in the lumbar enlargement, but the due action of this centre depends on cerebral (psychical) as well as reflex influences. Disease of the centre, or of the nerves leading to or from it, abolishes sexual action. The sexual reflex is, however, one of the cutaneous reflexes, and it shares the condition of these rather than of the muscle-reflex actions.[1]

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[1] This is sometimes well seen in locomotor ataxy. In rare cases of that dis-

The centre is probably double, and its action is impaired by interference with either half. When, by disease higher up, the connection with the psychical centres is interrupted, the sexual act cannot be perfectly performed. If the path from the controlling centre (p. 222) is unimpaired, the reflex sexual processes are not in excess, may even be diminished; but if the path from this controlling centre is also interrupted, the reflex sexual processes are in excess like the other superficial reflexes, and priapism results. If the reflex centre is partially diseased, the sexual act is imperfectly performed. The sexual centre is probably near that for the cremaster reflex, and from the latter we may, in some cases, gain information respecting the probable condition of the sexual centre, and in conjunction with the conditions just mentioned, of the sexual power. For instance, in locomotor ataxy, the cremaster reflex is rarely lost[1] without sexual power being lost or lessened also. Sexual power may, however, be lost before the cremaster reflex, perhaps because more readily impaired. Again, in a patient with extensive disease of the lower and middle part of the lumbar enlargement, greatest on the left side, in whom the cremaster reflex was present on the right side and lost on the left, sexual power was partially but not wholly lost.

Vaso-motor Centres.—The centres in the cord which influence the sympathetic and vaso-motor system of nerves are frequently affected in disease, and altered temperature, vascularity, and perspiration of limb result. In disease of the upper part of the cervical enlargement, especially in sudden lesions, hyperpyrexia may occur. But these symptoms at present are of little diagnostical importance, except when the disease is in the cervical region, and the vaso-motor change is conspicuous in the face. Then, if one side is affected, unilateral sweating and flushing are conspicuous, and are due to the fact that sympathetic fibres for the head arise in, or pass through the cervical cord. In the same cases, the movements of the iris are impaired: irritation of the cervical origin of the sympathetic causing spasm of the radiating fibres (dilatation), paralysis of the sympathetic causing their relaxation (contraction of the pupil). In many degenerative diseases of the cord

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ease, there is satyriasis and in such cases I have found a condition of extreme irritability of the cutaneous reflex actions.

[1] It must be remembered that the cremaster reflex is sometimes absent, in adults, apart from disease. The statement in the text applies to the cases in which there was such a general absence of the superficial reflexes as suggested a pathological loss.

the reflex action of the pupil to light is lost, the pupils being usually, but not always, small, but (as Argyll Robertson first showed) the pupils almost always contract if an effort of acccomodation is made. The reflex action is lost, but the associated action remains. When the pupils are small in these cases, they do not dilate on cutaneous stimulation, as they do in health (Erb). It is probable that these phenomena do not depend directly on the disease of the cord, but are due to an associated degeneration in the centres for the movements of the iris in the upper part of the pons.

In this survey of the more important functions of the cord, and their derangement, we have passed in review the chief symptoms which guide us in diagnosis. One or two others, however, remain.

Pain, referred to the spine, occasionally present in organic disease of the cord, is more frequent in disease originating in the meninges or bones. But the frequency with which spinal pain is present in abdominal, especially gastric, disease, and in neuralgic affections, lessens its diagnostic value when it exists alone. It is probably no exaggeration to say that of one hundred patients who complain of spinal pain, in ninety-nine there is no spinal disease. Moreover, in cases of organic disease, pain is far less frequent when the disease begins in the cord, than when it commences in the protecting structures. In meningitis, acute or chronic, spinal pain is frequent, and in organic disease of the bones of the vertebral column it is an almost constant symptom, and is combined with local tenderness. The same combination of local pain and tenderness is seen, however, in some cases of neuralgic pain, "rachialgia." The distinction between the two is that in organic disease there are indications either of displacement of the vertebræ or of changes in the cord.

A still more important group of pains are those which are referred to the parts to which the sensory nerves are distributed, and have hence been termed "excentric or irradiating pains." They are due to the irritation of the posterior nerve-roots in their passage through the intervertebral foramina, through the membranes, or through the posterior columns of the cord. Other similar pains are due, apparently, in some cases, to irritation of the sensory conducting tract higher up the cord.[1]

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[1] The latest physiological researches seem to show that the conducting tracts in the cord are not, as is commonly taught, entirely insensitive to local stimulation.

They may be dull pains, singularly resembling rheumatism, and constantly mistaken for rheumatism by the patients themselves and their medical attendants. The mistake is the more easily made because other symptoms suggestive of spinal disease may be inconspicuous, and the rheumatoid pains, in acute cases, may be accompanied by febrile symptoms, and in chronic cases may be influenced by weather, being much more troublesome in damp and cold than in fine and dry weather. In all cases, persistent rheumatic pains in the limbs should excite a suspicion of spinal disease, and watch should be kept for such symptoms as local loss of power, or alterations in reflex action. In other cases they are sharp darting pains, "like a flash of lightning and gone again," as they are often described by patients with locomotor ataxy, in which they are very frequent. Sometimes there is a momentary local stab, at others the pain seems to dart down the limb. The position in which these various excentric pains are felt—legs, trunk, or arms—depends (when the nerve-roots are irritated) upon the seat of the disease—in the lumbar, dorsal, or cervical region of the cord. Occasionally the irritation is felt, not as a sharp pain, but as a painful sense of tightness, as if a band were tied tightly around the limb or trunk—the "girdle-pain," as it is called. When there is transverse damage to the cord, at the lowest part of the healthy region there is a state of irritation of the sensory nerves, and this irritation (referred to the nerve-endings) causes the girdle-pain. When the nerve-roots are irritated by disease of the vertebræ, caries or cancer, the pain is very intense, and is especially increased by movement ("*paraplegia dolorosa*").

Whenever there are excentric pains there may be increased or diminished sensitiveness in the part to which the pains are referred. Spontaneous sensations are also common, the various feelings comprehended under the terms "numbness," "pins and needles," "furriness," "formication," and the like. The conditions in which they occur are various, but they should never be lightly passed over.

Spasm.—Muscular spasm is conspicuous in many cases of disease of the spinal cord. It depends on over-action of the motor centres. Primarily, perhaps, it is due to "diminished resistance" within them, but ultimately the functional action (and underlying nutrition) of these centres seem to be permanently altered. The motor centres are, as we have seen, both parts of the reflex centres, and the terminations of the path of voluntary impulse. Hence spinal spasm may be excited by peripheral impressions, or by attempts at voluntary motion. In some cases paroxysms appear to come on without excitation, especially during sleep. In sleep, however, the reflex action of the cord is very readily excited, and it is difficult to exclude slight reflex stimulation. As an acute symptom, spasm is almost confined to meningitis, and to some very rare forms of functional irritation. In meningitis the spasm is apparently reflex, produced by irritation of the nerves of the meninges. In chronic organic disease, spasm is usually a late symptom, of gradual development, and then its reflex character may often be distinctly traced. It occurs in cases in which muscle-reflex action is in excess, and this, as already explained, follows and indicates the occurrence of descending degeneration in the lateral columns (see p. 231). The increase in the muscle-reflex action is first manifested by an excess of the irritability which can be developed by tension (increased knee-jerk, foot-clonus), and this may be distinct at the end of a week or ten days. A further increase leads to occasional "stiffness" in the legs, especially at night; and ultimately there is developed a considerable degree of spasm, the condition known as "spasmodic or spastic paraplegia." Any peripheral impression, superficial or deep, pinching the skin, for instance, or sudden muscular tension, will then excite spasm. The attempt to elicit the foot-clonus may cause such muscular rigidity that no clonus can be obtained. In most cases the spasm is extensor in character, and evidently depends on the reflex mechanism which assists in maintaining extension of the legs in the erect posture. In health, when we stand, the muscles are in a state of balanced contraction, largely reflex, the afferent impulses being derived from muscles, possibly in part from joints. In spastic paraplegia a similar but more intense extensor contraction is excited by the same posture of the limb. Flexed, it may be supple, but extend it passively, and as soon as it is straight the muscles become rigid, and it cannot again be flexed except by considerable force. It is just as when a clasp-knife is opened, as soon as the blade is fully extended it becomes rigid. So this has been called "clasp-knife rigidity." Frequently the spasm fixes for the time both legs to the pelvis, and if one leg is lifted from the bed the other rises with it. The same extensor spasm occurs when the patient attempts to stand, and it often enables a patient to remain erect whose voluntary power would be insufficient for him to do so were he not aided by the spasm.

In some cases, especially during sleep, flexor spasm predominates, and the hip and knee joints become strongly flexed. On what the difference in the form of spasm depends we do not yet know. Spasm, especially flexor spasm, was formerly regarded as evidence of "chronic meningitis," because acute meningitis is accompanied by spasm. In many of these cases, however, there is no other evidence of meningeal disease.

Occasionally spasm occurs in violent paroxysms, first tonic, and then clonic, excited by slight peripheral impressions, and in some cases apparently spontaneous—the "spinal epilepsy" of Brown-Séquard. The resemblance to an epileptic paroxysm, however, is not close, and the quick clonic spasm depends on precisely the same conditions as the foot-clonus. The peripheral impression excites violent tonic spasm; as this is passing off, the tension on the imperfectly relaxed muscle is sufficient to develop clonic contractions, just as does the passive tension in the ordinary method of obtaining the foot-clonus, and so we have a series of quick clonic contractions succeeding tonic spasm. The effect is most conspicuous in the quadriceps extensor of the knee. Sometimes the initial tonic spasm is slight, and the spasm appears to consist entirely of clonic spasm.[1]

Thus these spasmodic phenomena indicate integrity of the reflex loops and functional over-activity of the reflex-centres. This over-action in chronic cases is the effect of disease above, in the lateral columns; the degeneration of their fibres extends down to the lower centres into the gray matter, and probably invades a structure in it which controls the muscle-reflex action (see p. 231). The gradual development of the over-action indicates that it is the result, in most cases, of changes consequent on this degeneration. Control being thus removed, the excessive reflex action gradually leads to what may be termed, if the expression is permissible, a functional hypertrophy of the centres, causing persistent and extreme spasm.

Simple rigidity of muscles, varying too little to be termed spasm, occurs also in some forms of disease of the cord, especially in cases of muscular atrophy (degeneration of the anterior cornua), and is due to simultaneous (or, according to Charcot, preceding) degeneration in the lateral columns. Persistent shortening (contracture) occurs also in the antagonists of paralyzed muscles, but now and then as a result of over-action from central disease. As a consequence of this the knees may become flexed or the heels drawn up. The latter form of rigidity, dependent on a primary over-action, is always associated with more or less general spasm in the limb—an important distinction from the condition in which such shortening is due to the paralysis of the opponents of the contracted muscles. Persistent contracture of the gastrocnemii, as part of "spastic paraplegia," is sometimes seen in adults, but is more common in children.

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[1] It is necessary to mention that the term "spinal epilepsy" has been misapplied in France to the foot-clonus.

III.—INDICATIONS OF POSITION OF DISEASE: ANATOMICAL DIAGNOSIS.

We may now consider, briefly, how the symptoms which we have studied are grouped in diseases of different regions of the cord. The various symptoms, and their significance, have already been considered in detail, so that it is necessary only to mention them here. Some lesions of the cord affect certain structures (white columns or gray matter) in a considerable vertical extent, the other structures being normal. Such affections have been called "system diseases." Others, again, are very limited in their vertical extent, and have been termed "focal" lesions. The latter may be limited to one structure, or may extend through a considerable transverse extent, even through the whole thickness of the cord—"total transverse lesions." The lesions which affect certain structures only, whether extensive system diseases or limited focal diseases, are called "partial lesions," and it is convenient to commence with these.

1. *Antero-lateral White Columns.*—Disease of the antero-lateral white columns causes loss or impairment of voluntary power below the lesion, descending degeneration in the pyramidal tracts, and over-action of the lower centres, especially of those concerned in the muscle-reflex processes. This over-action may be manifested only as excess of the myotatic contractions, or it may increase to spasm and rigidity—spastic or spasmodic paraplegia. There is no wasting unless the degeneration, extending from the lateral columns, invades the motor nerve-cells in the anterior cornua. Then we have a combination of spasm and wasting in which, if the cornual degeneration proceeds, the spasm and rigidity may lessen as the nerve-cells suffer. In disease limited to these columns (at any rate, when the disease is limited to the pyramidal tracts) there is no loss of sensation, or inco-ordination. These symptoms of "spasmodic

paraplegia" may arise from a primary degeneration in the lateral columns, limited thereto; but such cases are rare, and in the majority the disease is a focal lesion more or less extensive at some level in the dorsal or cervical cord, and the degeneration in the lateral columns is secondary. The evidence of the latter form is afforded (1) by the circumstance that the symptoms came on, in the first instance, suddenly or rapidly, primary sclerosis being gradual in onset, and a lesion which occurs in short time is always "focal;" (2) the evidence which may generally be discovered that there has been at some time, or is in some region, damage which extends beyond the lateral columns. The proof of this is the implication of sensation, or the interference, at the level of the lesion, with the central functions of the cord. We are only justified in suspecting a primary sclerosis of the lateral columns when we can find no such evidence or history of a wider focal lesion, and when the affection came on very gradually; and we can only feel *sure* that it exists when the arms become affected in the same manner as the legs, and after them. We must remember also that descending lateral sclerosis, with secondary spasmodic phenomena in the limbs, may also result from damage to the motor tracts above their decussation—in the medulla, the pons, or the motor parts of the cerebral hemispheres. It occasionally results from bilateral damage to the surface of the brain during difficult birth.

Certain lesions may damage the motor tracts slightly, and impair conduction in a peculiar way, apparently rendering it unequal in different fibres. As a consequence, the muscular action is disproportionate in different muscles, and instead of a balanced co-ordinated movement, we have an unbalanced jerky movement. This is seen especially when irregular islets of sclerosis affect the cord—disseminated or insular sclerosis; and according to the researches of Charcot it appears that this irregular conduction is the result of the unequal wasting of the medullary sheaths, the axis-cylinders remaining. A precisely similar symptom may result from pressure on the motor tract—as by a growth. Not rarely this "disseminated" or "insular" sclerosis, in one region, is combined with a system-degeneration in another. An occasional combination, for instance, is the jerking movement (from cervical insular sclerosis) in the arms, and ataxic inco-ordination in the legs (from lumbar posterior sclerosis). Or with the jerky inco-ordination in the arm there may be weakness with spasm in the legs from lumbar lateral sclerosis. In the latter case it is probable that the lateral sclerosis is simply "descending," the result of the damage to the pyramidal tracts higher up by the insular sclerosis. It must be remembered that insular sclerosis sometimes causes merely loss of power, and equal, not irregular, impairment of conduction, especially when it occurs in the dorsal region. In such a case we may be unable to distinguish its symptoms from those of a diffuse, widely-spread degeneration.

2. *Posterior Columns.*—In disease of the posterior columns there is interference with co-ordination without loss of power; excentric pains, impaired sensation, and diminution of reflex action, in consequence of the implication of the sensory roots. All these symptoms depend on disease of the postero-external columns (posterior root zone).[1] Disease of the postero-median columns give rise to no known symptoms.

The posterior columns may be damaged by any pathological process; they are frequent seats of primary degeneration (sclerosis), the condition which constitutes the common form of locomotor ataxy. The symptoms of this disease usually present the following order—loss of the myotatic contractions, especially of the knee-jerk, pains, inco-ordination, diminution of sensation, loss of sexual power, and of the reflex-action from the skin (which may in the early stage be increased), affection of the sphincters and occasionally interference with the nutrition of bones and joints.

There is no loss of motor power or wasting as long as the disease remains limited to the posterior columns. It may, however, extend forwards into the anterior cornua, causing muscular artrophy and weakness to be conjoined with the ataxy. Or the lateral columns may be affected at the same time as the posterior: we then have weakness as well as ataxy, but no wasting. The disease of the lateral columns causes, as I have just stated, increase of the muscle-reflex action, and this increase may thus co-exist with inco-ordination, the damage to the posterior roots being slight, perhaps absent, in these cases (see p. 236). Thus we may have the anomaly of ataxy with actual loss of power, excess of the knee-jerk instead of its loss, and the front-tap contraction obtainable, and even the foot-clonus.

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[1] It is to be noted, however, that recent researches, especially those of Pierret and Déjerine, have shown that there is frequently an independent degeneration of the peripheral sensory nerves. The extent to which the symptoms of the disease depend on this is at present undetermined. It does not appear to be invariable, and the effect is probably the same, whether the nerve-fibres are damaged at the periphery or in the posterior columns of the cord.

Associated with primary degeneration of the posterior column are certain other symptoms due to simultaneous degeneration elsewhere. Of these the most important are loss of reflex action of the iris to light, which is extremely common, loss of accommodation, which is rare, and optic nerve atrophy, which is occasional.

An important fact to remember, regarding the posterior columns, is their proneness to degenerate: they recover less readily, and degenerate more readily, than any other structure in the cord. A lesion in one spot may set up a degeneration which ultimately involves them in their whole extent. Damage affecting the whole thickness of the cord may pass away from the rest, and persist in the posterior columns, and even spread there. In such a case we have ataxy succeeding loss of power. Movement returns, but without co-ordination.

3. *Anterior Cornua.*—The anterior cornua contain the motor nerve-cells, which, as I have said—(1) influence the nutrition of the motor nerve-fibres proceeding from them, and consequently that of the muscles; (2) constitute a link in the path of the voluntary impulse from the brain to the muscles; (3) form part of the reflex loop, probably also of the reflex centre, with which the muscles are connected.

Hence we have, as the result of disease of the anterior cornua—(1) degeneration of the motor nerves and wasting of the muscles; (2) loss of voluntary power, *i.e.*, paralysis of those muscles; (3) interference with, or arrest of, all the reflex actions in which these muscles take part.

The extent of these symptoms, whether they are unilateral or bilateral, affect many muscles or few, will depend strictly on the extent of the disease in the spinal cord.

Of the three symptoms, the muscular wasting is incomparably the most important. Paralysis may result from disease elsewhere in the motor tract, *e.g.*, disease of the lateral column higher up. Loss of reflex action may depend on disease elsewhere in the reflex loop, *e.g.*, disease of the sensory fibres in or outside the cord. But muscular wasting is due only to a lesion of the motor cells, or to a lesion of the nerves cutting the muscles off from the influence of these cells. In most cases we are able to exclude the latter (by indications presently to be mentioned): the state of muscular nutrition comes thus to be of the highest importance as indicative of the state of the anterior cornua of the cord. To learn their condition further, we ascertain the electrical excitability of the nerves and muscles, according to the principles already laid down.

Whenever we find wasting, and infer that there is disease of the anterior cornua, we have next to observe whether the weakness and wasting are proportioned, *i.e.*, whether the weakness is only such as the affection of the gray matter will account for, or whether it is in such excess as to indicate other disease in the motor tract.

In acute diseases of the anterior cornua, paralysis precedes wasting. The sudden interruption of the motor path causes immediate loss of power. Wasting of muscles succeeds the degeneration of the motor nerves, occurring a week or ten days after the loss of power. In chronic diseases the wasting and weakness come on together.

Chronic disease of the anterior cornua is often combined with disease of the lateral (pyramidal) columns similar to the descending degeneration. Charcot believes that in these cases the degeneration in the lateral column is primary, its symptom, muscular rigidity, preceding the symptom of the cornual disease, muscular wasting, and he terms the affection "lateral amyotrophic sclerosis." It is possible, however, that this position will need reconsideration, and that the degeneration in the lateral columns is, sometimes at least, secondary to, or simultaneous with, the disease in the cornua. It often extends, however, beyond the fibres related to the degenerated cornua, and so may cause weakness and spasm in the limbs below the seat of the muscular atrophy. Thus we have wasting in the arms, and weakness with spasm in the legs, and even, as I have seen, wasting in the shoulder muscles, and weakness without wasting in the hands.

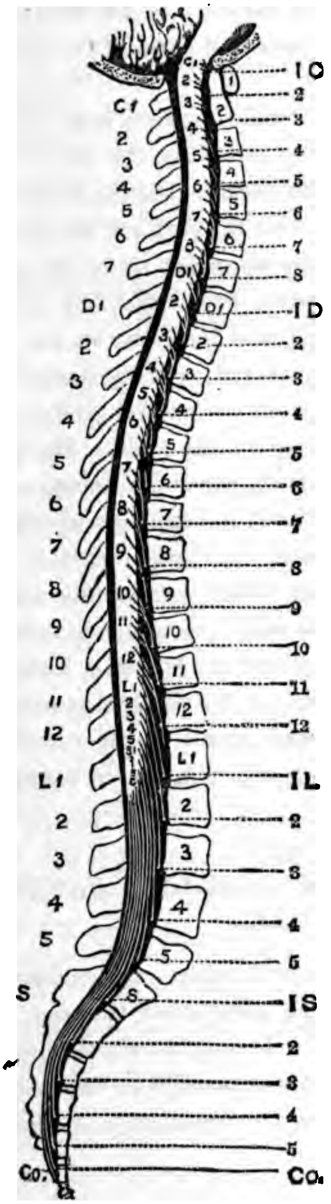
A lesion of the anterior cornua never, *per se*, affects sensation. Acute lesions in this situation may, however, disturb adjacent sensory parts (posterior cornu or sensory tract), and so cause "excentric" pains, often rheumatic in character. Actual loss of sensation, with wasting, points, especially if irregular in distribution, to damage to the nerve-roots outside the cord, and therefore to disease of the meninges rather than of the cord itself.

4. *Unilateral Lesions.*—These interfere with the conduction of the motor impulse on the same side as the lesion, and so cause weakness in one side, "hemi-paraplegia" or "spinal hemiplegia," and often descend-

ing unilateral sclerosis with its symptoms in the affected leg. Whether there is weakness of the other leg (if the lesion is strictly unilateral) will depend on the number of pyramidal fibres, which in the "direct pyramidal tract," have not decussated at the seat of the lesion; and this varies, as we have seen, in different individuals. It must be remembered, however, that in unilateral lesions, the opposite half of the cord is often slightly damaged, and the symptoms are therefore rarely limited to one leg. Sensation is affected on the opposite side to motion, but not quite up to the level of the lesion, because the decussation of the sensory tract is not immediate, but occurs a little above the place at which the nerves enter the cord. Sometimes, however, sensation is affected on the same side as motion. This is often the case when the paralysis affects the leg only, and probably the lesion is, in such cases, so placed as to impair the sensory fibres before their decussation.

5. *Total Transverse Lesions.*—A total transverse lesion of the cord, at any level, however limited in vertical extent, separates all parts below the lesion from the brain, and hence, so far as will and perception are concerned, produces the same effect as if the whole of the cord below the lesion were destroyed. A section across the cord in the middle of the cervical enlargement, for instance, paralyzes all parts below the neck with the exception of the diaphragm. Hence the extent of the paralysis indicates only the upward limit of the lesion. This is also indicated by the position of the girdle-pain and radiating pains, or zone of hyperæsthesia, which are due to the irritation of the sensory roots in the lowest part of the upper segment—an important indication when the lesion is in the dorsal region, where the precise limitation of motor weakness may be recognized with difficulty.

It is important, however, to know the symptoms which occur in disease at different levels. These are shown in the accompanying diagram and table (Fig. 32), and may be understood from the following description. The indication of the upper level of the lesion is afforded by the loss of the motor and sensory functions, shown in the first two columns. The lowest nerves supply the anus and perineum. The nerves which supply the skin and muscles of the leg and foot arise from the 1st to the 3d sacral nerves, and are damaged by a lesion involving the lower part of the lumbar enlargement. We must remember, however, that the skin on the inner side of the leg is not supplied from this source, and so may escape when the outer part of leg and back of the thigh have lost sensation. In the middle of the lumbar enlargement, we have the nerves arising which enter the lumbo-sacral cord, and these are probably destined for the flexors of the knee, and for the hip muscles which are supplied by the sacral plexus, the glutei, the quadratus, and gemelli, and the skin of the lower part of the gluteal region. These parts then will be paralyzed by disease in the middle of the lumbar enlargement, while the muscles and skin in front of the thigh are unaffected. The latter suffer when the disease affects the upper part of the lumbar enlargement, the origin of the anterior crural (rectus, etc.), and obturator (adductors). The skin on the upper and outer parts of the thigh loses sensibility, with the part adjacent to the scrotum, and in the groin, only when the disease damages the highest part of the lumbar enlargement, from which the first three lumbar nerves arise, and then the flexors of the hips become paralyzed. In proportion as the disease is higher in the dorsal region, we have the symptoms ascending higher up the trunk, and marking accurately the height of the lesion by the loss of cutaneous sensibility, and by the impairment—first, of the abdominal muscles, and then of the intercostal muscles. The umbilicus corresponds to the 10th dorsal nerves, and the ensiform area to the 6th and 7th. When the disease reaches the lowest part of the cervical enlargement (the 1st dorsal nerves), we have the first symptoms in the upper extremity; but these are not, as might be expected, in the muscles moving the shoulder joint, but in the hand. The first numbness is complained of in the little finger, and the first weakness is in the intrinsic muscles of the hand. Ascending higher, the symptoms pass up the arm with some uniformity, and without respect to nerve distribution. When the middle of the cervical enlargement is reached (the 5th, 6th, 7th cervical) the shoulder muscles and the serratus magnus become paralyzed, and there is general loss of power and sensation and anæsthesia. Above the level of the 6th pair, the trapezius and sterno-mastoid become somewhat weakened, for the fibres of the spinal accessory which supply them undoubtedly arise in part from this region of the cord. At the 4th and 5th cervical the lower part of the neck becomes anæsthetic, and the diaphragm ceases to act. Here our localization might cease, for total transverse lesions at this spot necessarily cause death. For a little time the sterno-mastoids and scaleni can get air into the chest, but not in sufficient amount to maintain life for more than a few days. But limited lesions may occur higher up, and then we have com-



MOTOR.	SENSORY.	REFLEX.
St.-mastoid Trapezius	Neck and Scalp	
Diaphragm	Neck and Shoulder	
Serratus		
Shoulder	Shoulder	
Arm mus.	Arm	Scapular
Hand (ulnar n. lowest)	Hand	
Intercostal Muscles	Front of Thorax	Epigastric
	Ensiform area	
Abdominal Muscles	Abdomen (Umbilicus 10th)	Abdominal
	Buttock, upper part	
	Groin and Scrotum (front)	Cremasteric
Flexors, hip	outer side	
Extensors, knee	Thigh front	*Knee-jerk*
Adductors		
hip	inner side	
Abductors	Leg, inner side	Gluteal
Extensors(?)	Buttock, lower part	
Flexors, knee (?)	Back of Thigh	
Muscles of leg moving foot	Leg and Foot except inner part	*Foot-clonus*
		Plantar
Perineal and Anal muscles	Perineum and Anus	
	Skin from coccyx to anus	

FIG. 32.—DIAGRAM AND TABLE SHOWING THE APPROXIMATE RELATION TO THE SPINAL NERVES OF THE VARIOUS MOTOR, SENSORY, AND REFLEX FUNCTIONS OF THE SPINAL CORD. (*From anatomical and pathological data.*)

plete powerlessness of the muscles moving the head, upper part of trapezius and sterno-mastoid, and other muscles attached to the occipital bone, and interference with sensation in the neck and parts of the head, which are not supplied by the 5th nerve.

The extent downwards of the lesion, its vertical extent, is thus not indicated by the impairment of its conducting functions, the motor or sensory paralysis; and to learn *this* we have to examine the functions of the cord as a central organ, and to ascertain how far they are impaired in the paralyzed region—to examine especially muscular nutrition and reflex action. The state of muscular nutrition and irritability indicates how far the anterior cornua are impaired, and the latter shows, as I have explained (p. 240), whether they are involved in the primary lesion or are affected secondarily. The relation of the several groups of muscles to the cord is shown in the first column of the table. The integrity of reflex action indicates the integrity of the reflex loops, and the study of the superficial reflexes of the trunk is especially instructive in this respect. The series of reflexes, and the relation of each to the cord, are shown in the third column of the table; the myotatic contractions are printed in italics in the position which corresponds to the centres, the influence of which is essential for their production. Excess of superficial reflex action indicates withdrawal of the cerebral controlling influence of the reflex centres, and marked excess of the muscle-reflexes suggests the existence of a descending degeneration in the lateral columns.

IV.—INDICATIONS OF NATURE OF DISEASE: PATHOLOGICAL DIAGNOSIS.

The last part of our subject remains for consideration—the elements of the pathological diagnosis, by which, having ascertained the seat of the lesion, we endeavor to learn its nature. To do this, we attend, first, to the way the symptoms come on and develop; secondly, to the position and distribution of the lesion; thirdly, to any causal or associated conditions which may be present.

We may group the primary morbid states into the following forms:—

(*a*) Vascular lesions; rupture of vessels, causing hæmorrhage; occlusion of vessels, from thrombosis or embolism (the latter being very rare).

(*b*) Inflammation; "myelitis," acute or chronic, the former causing softening. It is common to call all forms of softening "myelitis;" we do not yet know how far they are originally inflammatory, or are set up, as in the brain, by vascular occlusion. Some chronic inflammations are not attended by softening.

(*c*) Degeneration and "sclerosis," in which the nerve-fibres waste, and the connective tissue (neuroglia) overgrows.

The term "sclerosis" is inaccurate etymologically, since the part altered by increase of connective tissue elements is often softer than normal, but the term is firmly established. In some cases the change appears to commence in the nerve-fibres, in others in the connective tissue. Some forms of degeneration pass by graduations into chronic inflammation (here as elsewhere), and the term "chronic myelitis" is sometimes applied to the slow degenerative forms. On the other hand, the condition of "sclerosis" may result from inflammation. The term is thus used in two senses, to indicate a pathological process, and a pathological condition which may result from more than one morbid change. Here the term will be used, when unqualified, to designate the process.

It is most important to remember that degeneration and sclerosis are not identical terms. Primary degeneration of the nerve-fibres is followed, after weeks or months, by overgrowth of the interstitial tissue, which takes their place.

(*d*) Pressure from without, by inflammatory swelling of meninges, or by displaced bones, or by growths.

(*e*) Growths in the cord itself.

I. We have first to consider how far these several lesions can be distinguished by their onset, *i.e.*, by the time occupied in their development to a considerable degree of intensity. According to this, we may divide them into five classes: those in which the onset is *sudden*, instantaneous or nearly so; *acute*, occupying a few hours to a few days; *subacute*, developing in one to six weeks; and lastly, the *chronic* cases.

which may be divided into those occupying six weeks to six months, and those ocupying six months and upwards in their onset.

I have endeavored to show the common relation of the lesions to these several courses in the following table :—

	Onset.	
	SUDDEN (*few minutes*)	Vascular lesions.
	ACUTE (*few hours or days*)	
Pressure or Growths	SUBACUTE (*one to six weeks*)	Inflammation (myelitis).
	CHRONIC (*six weeks to six months*)	Degeneration (sclerosis).
	VERY CHRONIC (*six months and upwards*)	

A lesion of sudden occurrence, developing symptoms in the course of a few minutes, is almost always vascular; commonly hæmorrhage, perhaps vascular obstruction. But a vascular lesion may occupy a somewhat longer time in development—a few hours or days. In acute and subacute inflammation the symptoms come on in the course of a few hours, a few days, or a week or two. Chronic inflammation occupies from a few weeks to a few months. Degeneration, in which there is no adequate evidence of any inflammatory process, occupies many months, or it may be years. The symptoms produced by growths or simple pressure (traumatic causes excluded) are never sudden or very acute, and rarely, if ever, very chronic, the time occupied by the development of the symptoms varying, according to the nature of the cause, from a fortnight to six months.

It is necessary to consider, however, not merely the whole time occupied by the development of the disease, but also the uniformity of its course. Two or more morbid processes may concur. An initial myelitis, for instance, may lead to a secondary degeneration; and, on the other hand, in degenerated tissues sudden vascular lesions occasionally occur. Pressure often produces local myelitis, which may be very acute in its development. Cancer of the vertebræ, for instance, usually causes rapid myelitis. The whole course of the disease must be ascertained before an inference is drawn.

The onset and course of the symptoms thus sometimes enable us to decide at once that a lesion is of a given character, as that one which occurs instantly is vascular, or that one which takes years for its development is degenerative. More frequently they enable us to exclude certain morbid processes, and to restrict the possible lesion to two or three forms. For instance, a lesion which comes on in the course of a few hours must be either vascular or inflammatory. Between these we have to decide by attention to other indications.

II. In actual diagnosis it is convenient to consider next the indication afforded by the position and distribution of the disease. We consider what diseases occur in this situation, and then which of them have the mode of onset which has been ascertained. As I said at the outset, this indication is only to be employed in subordination to a careful study of the mode of onset and course.

The gray matter of the cord is the most frequent seat of hæmorrhage. Either gray or white substance may be the seat of inflammation or of degeneration. In young children inflammation is much more common in the gray matter than in the white. Pressure or growths usually first affect the white columns, but may afterwards involve the gray matter.

The affections called "system-diseases," in which one system of structure is affected through a wide vertical extent of the cord, are commonly degenerative in nature: such are lateral sclerosis, posterior sclerosis (locomotor ataxy), the change in the anterior cornua which leads to progressive muscular atrophy (anterior cornual degeneration). These processes probably begin in the nerve elements. On the other hand, lesions which have a limited vertical extent—"focal lesions"—are commonly the result of processes which may be either acute or chronic, but begin outside the nerve elements, in the connective tissue, vessels, etc. Such are hæmorrhages, foci of myelitis, spots of "insular" sclerosis, growths, and pressure from without.

But this distinction cannot be employed except after due consideration of the mode of onset. Scattered acute focal lesions, for instance, may be widely distributed in the same structure, and produce symptoms limited to certain functions, but of wide extent, and simulating—indeed, constituting—a "system-disease." Thus I have seen subacute symmetrical myelitis of the anterior cornua in the lumbar and cervical enlargement cause paralysis and atrophy in all four extremities, the upper parts of the limbs being normal. Again, a small focal lesion may be limited to one structure, and cause symptoms confined to one function. Thus we may have an anterior cornual myelitis, or a columnal myelitis, lateral or posterior, giving rise to limited symptoms—local muscular atrophy, unilateral paralysis, or local ataxy. Lastly, many "focal lesions" may give rise to secondary system-degenerations. A focus of myelitis in one lateral column may cause descending degeneration in the whole lateral column below, with its attendant spasmodic symptoms. Indeed, so true is this, that, as we have seen, lateral sclerosis is more often secondary to a limited focal lesion than primary. In all these cases, however, attention to the mode of onset will prevent error.

The combination of mode of onset with seat of lesion sometimes helps us in a more direct manner, especially in the case of growths and compression. The characteristics are their limited vertical extent, gradual onset, and slow invasion of parts adjacent to that first affected, on the same level; one leg, for instance, is affected, and then the other.

Indication of disease outside the cord, irritation of certain nerve roots, causing severe local pain, often precedes the symptoms of compression and is an important aid to diagnosis. It shows the existence of a morbid process outside the cord before the cord is involved. But we cannot use even this indication except in dependence on the mode of onset. A disease, as a growth outside the cord, may not only compress the cord, and cause slow loss of power; it may excite inflammation and cause rapid palsy.

There is a rare form of paralysis, in which the functions of the cord are progressively impaired from below upwards, until, in the course of a few days, death results from interference with respiration. In these cases of "acute ascending paralysis," as they are called, no lesion of the cord has usually been discovered, and their exact nature is unknown.

III. The last element in the pathological diagnosis is the detection of any influence which can be regarded as the cause of the disease in the spinal cord, or any associated condition which may indicate an active morbid process. We have seen that the mode of onset may help us to limit the disease to certain possible forms of lesion: the distribution of the affection may render it probable that it is one or other of these forms; and the detection of a cause of disease of the spinal cord, and the knowledge of the lesions which that cause produces, may help us to fix the nature of the lesion still further. It is important, therefore, in diagnosis, to be aware of the several effects of the common causes of spinal disease.

1. *The Age of the Patient.*—In young children acute myelitis is the most frequent lesion, and the gray matter is usually the chief seat of the disease.

2. *The State of the Vascular System.*—The conditions which favor hæmorrhage are of far less diagnostic value with regard to the spinal cord than with regard to the brain. Conditions of mechanical congestion—heart disease, emphysema, etc.—favor degenerative changes and also, probably, thrombosis. The state of the vascular system which is associated with chronic kidney disease undoubtedly favors degenerative changes in the cord, the occurrence of which has been demonstrated by Sir William Gull and Dr. Sutton.

3. *Scrofula* commonly causes spinal disease by leading to disease of the bones of the spinal column; and the evidence of this, local tenderness or irregularity of the vertebral spines, or actual curvature, is of the highest diagnostic importance, and careful and repeated examination of the bones should never be neglected in cases of obscure spinal disease. There is, perhaps, no error in diagnosis which is more frequently made, or which results in graver errors in treatment, than the non-recognition of disease of the spinal bones. It is important to remember that the damage to the cord may occur before the signs of bone disease are distinct: hence the importance of *repeated* examinations.

In bone disease the cord suffers in at least four different ways:—(*a*) By pressure from the inflammatory swelling of the bone or inflammatory products without curvature. The effects of the pressure may lessen as the curvature comes on. (*b*) By pressure in consequence of the displacement, the bony canal being narrowed by the angular projection of the bodies over which the cord is stretched. (*c*) By secondary chronic inflammation, with thickening, of the dura mater and tissue outside it, compressing the cord. (*d*) By inflammation extending through the membranes to the cord, or set up in the cord by the compression. Hence we cannot, because we find evidence of bone disease, immediately conclude that the cord is pressed upon by the displaced bone. We must investigate the mode of onset of the symptoms and their character, and infer from these the character of the disease of the cord according to the rules now given. The most common mechanisn is compression by products of inflammation outside the dura mater, and by the thickening of this membrane.

In recognizing bone disease it must be remembered that not only may there be no angular curvature until long after the cord has suffered, but there may not even be irregularity of the vertebral spines. There are often pain and local deep tenderness to be elicited. In the resulting paraplegia the excess of the cutaneous reflexes is often an early and conspicuous symptom, and spots of anæsthesia at the level of the bone disease, due to pressure on nerves, may sometimes be found, and give important help in diagnosis.

4. *Syphilis.*—The methods by which syphilis causes disease of the cord, which are universally recognized, are—

(*a*) The growth of syphilomata springing from the connective tissue, the membranes, or tissue in the fissures, and invading the cord. In these cases we have symptoms varying in character according to the position of the growth, and similar to those produced by other limited lesions, but always of gradual onset.

(*b*) By chronic meningitis, with thickening and pressure on the nerves, and sometimes on the cord also. The characteristic symptoms depend upon the damage to both motor and sensory nerves, that of the former cutting off the muscles and peripheral nerves from the influence of the motor nerve-cells, and hence causing muscular atrophy, very similar to that due to disease of the gray matter, but differing by its association with scattered areas of diminished sensitiveness of the skin. The interference with the reflex loops abolishes reflex action in the part; but if the damage is confined to the upper part of the cord and the cord itself is pressed upon, there may be an excess of the reflex action on the lower part.

(*c*) Syphilitic disease of vessels may *probably* lead to acute softening, similar to that in the brain. Syphilitic subjects may become suddenly paraplegic, and it is possible that it is by this mechanism, although the fact does not at present rest on post-mortem evidence.

All the above lesions originate in the adventitial structures. There is, however, a considerable mass of evidence to show that (*d*) diseases which originate in the nerve elements and neuroglia, more or less inflammatory or degenerative in character, may be a late effect of syphilis. I have found, for instance, disseminated foci of chronic myelitis, affecting chiefly the periphery of the cord, throughout the dorsal region, in a syphilitic woman, and two similar cases have been observed by Pierret. It is probable that this form of chronic myelitis is usually syphilitic.

Symptoms of acute myelitis sometimes occur in syphilitic subjects, and the myelitis has been regarded as due to the syphilis, but the evidence of this is at present insufficient.

The majority of cases (about 70 per cent) of locomotor ataxy, primary posterior sclerosis, occur in individuals who have had syphilis many years before.[1] Anterior cornual degeneration (progressive muscular atrophy) sometimes occurs after constitutional syphilis, and so also do the symptoms associated with sclerosis of the lateral columns. In these cases of degenerative disease it does not appear, as far as we can tell, that the anatomical process presents any recognizable difference from that which occurs as the result of other causes; and it is possible that the influence to syphilis, although effective, may not be direct, *i.e.*, the disease is due, in part at least, to the preceding syphilis, although it is not syphilitic in nature. One consequence of syphilis may be to cause a neuropathic tendency, in which these diseases are gradually developed.

Although not strictly a fact of etiology, I may mention that the result of treatment often affords an important corroboration of the diagnosis of syphilitic disease. If symptoms, which we have reason to suppose are due to syphilitic disease, improve rapidly when iodide of potassium or mercury is given, the diagnosis is strongly corroborated.[2] But the converse of this is not equally true. A disease may be due to syphilis, and no improvement be obtained from specific treatment. It must be remembered that as regards (*a*), (*b*), and (*c*), the syphilitic disease causes symptoms by producing changes in the nerve-elements, softtening, degeneration, etc., which are not in any way syphilitic, but are such as would result from adjacent disease of any other nature. Under some conditions (of intensity, duration, etc.), the recovery of the nerve-tissue may be impossible, even though the syphilitic adneural disease be completely removed. Further, the diseases of the last class (*d*) are not, except in the earliest stages, benefited to any marked extent by anti-syphilitic treatment.

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[1] See "Syphilitic Neuroses," "Brit. Med. Journal," March, 1879. A similar statement had been expressed before by Fournier, and since has been made by Vulpian and Erb. I have given their statements in a paper on the subject of "Syphilis and Locomotor Ataxy," in the "Lancet" for January, 1881. It is not suggested that syphilis is the cause in this proportion; in some the coincidence of the two diseases may be accidental. The facts seem to justify the assertion that half the patients would not suffer from ataxy had they not previously suffered from syphilis.

[2] Always provided the symptoms are not such as tend to lessen spontaneously. I do not mention this exception in the text, because, important as it is, it has less application to the syphilitic disease of the spinal cord than to those of the brain.

5. *The exciting causes* of disease of the spinal cord sometimes afford diagnostic indications. Exposure to cold may cause acute symptoms commonly due to inflammatory softening—sometimes focal, sometimes diffuse; and in the latter case often accompanied by symptoms of meningitis. It may also cause hæmorrhage. It is especially effective in women at the menstrual period. Repeated exposure may lead to degeneration, especially in the gray matter.

Acute specific diseases, as typhoid fever, are occasionally followed by spinal symptoms, due to changes which are probably of the nature of subacute inflammation. It is very common for a patient, after typhoid fever, to suffer for a long time, sometimes permanently, from weakness of the legs; and occasionally during the course of the disease acute symptoms, as those of anterior cornual myelitis, may occur.

Sexual excess is a more common cause of transient functional weakness than of organic disease.

Traumatic influences are frequent causes of cord disease. The cord may be directly pressed upon and damaged by displacement or fracture of the vertebræ, or a severe concussion may be followed by slow paralysis at an interval of a few days or weeks. In such a case, occurring after a railway accident, I have found numerous minute foci of chronic inflammation, most abundant in the gray matter. Sometimes a still longer interval elapses between the injury and the paralysis. In such cases a growth or patch of sclerosis appears to be set up by the injury, although years may pass before the symptoms reach a considerable degree of intensity.[1]

These, then, are the chief etiological facts which, taken in conjunction with mode of onset and distribution, enable us to form an opinion regarding the nature of the lesion.

To sum up: In examining a case of disease of the spinal cord, the method should be briefly as follows:—First, endeavor to ascertain the exact seat of the lesion; note how far the several conducting functions of the cord are impaired, and the highest level of the impairment; then ascertain the condition of the central functions, especially muscular nutrition and irritability, and reflex action, first in the part below the level at which conduction is impaired, and secondly at the supposed level of the lesion; and in this way you may infer, without much difficulty, what is the extent of the lesion transversely and vertically. In the next place endeavor to ascertain its nature by considering—first how the symptoms came on and developed; secondly, which of the lesions having this mode of onset and development occur in the region affected; and thirdly, which of them are produced by the cause or causes to which the disease is apparently due.

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[1] An instructive instance of the way in which the results of an injury of the head may cause both growths and arterial disease, and, years later, both chronic and acute symptoms, will be found recorded in the author's "Medical Ophthalmoscopy," Case 4, p. 248.

This process of diagnosis may seem somewhat elaborate, and, no doubt, a practised observer does not always consciously go through it. But, in most cases, if he wish to avoid error, he goes through it unconsciously, and no step can be with safety dispensed with. We may thus, in almost all cases, arrive at an exact diagnosis of the seat of the disease, and, in a large number of cases, of its nature also. There are, however, some cases with respect to which the diagnosis of the nature of the lesion can be approximate only, although we can always limit it to one or two possibilities.

It will be observed that I have said nothing of "anæmia of the cord," of "hyperæmia of the cord," or of "reflex paralysis." In current descriptions of the symptoms of these conditions, I cannot help thinking that a vigorous scientific imagination has contributed much more than observation has supplied. The only practical knowledge of the effects of anæmia and hyperæmia of the cord, is, that they seem capable of causing such disturbance of the sensory structures as reveals itself in subjective sensations of tingling, pins and needles, and the like, and perhaps also some impairment of motor conduction. A large number of authorities here and abroad are sceptical as to the existence of such a condition as "reflex paralysis," *i.e.*, a paralysis due to the effect on the centre of some peripheral irritation, disappearing when this was removed. Although our modern knowledge of the various phenomena of inhibition and reflex action renders such a paralysis *à priori* even probable, it is certain that the theory has been extensively misapplied.

Spinal Meningitis.—The object of this lecture has been to explain the principles of the diagnosis of diseases of the cord itself. But it may be well to allude briefly to the diagnosis of spinal meningitis. Of acute meningitis I need say little. The acute symptoms, spinal pain, and severe spasms, are well known. Chronic spinal meningitis, however, is a disease regarding which current opinion has curiously changed during the last fifteen years. A large number of symptoms were assigned to chronic meningitis which we now know have nothing to do with that pathological state. I have mentioned that to it the symptoms of weakness with chronic spasm, "spasmodic paraplegia," were ascribed. But we now know that these are due to alterations within the cord, and are independent of any meningitis. The only symptoms which are usually due to this condition are those which result from the involvement of the nerve-roots in their passage through the diseased membrane. The roots are irritated by the adjacent inflammation. The meninges frequently become much thickened, and by this thickening the nerve-roots are often greatly damaged. The irritation affects first the sensory roots, causing "excentric" pains and hyperæsthesia, to which are often added areas of anæsthesia here and there, due to the greater damage of some nerve-roots. The affection of the motor roots causes symptoms similar to those of disease of the anterior cornua, but very irregular in distribution. The peripheral motor nerve-fibres, cut off from their motor cells, degenerate, and the muscular fibres waste, and present electrical reactions which vary according to the rapidity of the morbid process. Sometimes the nutrition of the skin suffers. There is frequently, in addition, pain in the back, from the lumbar to the cervical region, sometimes severe between the shoulders, and accompanied sometimes with rigidity of the vertebral muscles.

The chief conditions with which chronic spinal meningitis may be confounded are posterior sclerosis (locomotor ataxy) in which the sensory nerve-roots are implicated, and anterior cornual degeneration (progressive muscular atrophy). From the former it is distinguished by the absence of ataxy, from the latter by the irregular distribution of the symptoms, and from both by the existence of limited areas of anæsthesia, and of extensive spinal pain. It must be remembered that inflammation often affects the substance of the cord as well as the meninges, or the cord may be pressed upon by the thickened membranes, and so mixed symptoms may result.

A word on the subject of the nomenclature of diseases of the spinal cord. If we wish to obtain clear ideas, it is essential to use terms, where we can, which shall be pathological, and which shall be at once simple and descriptive. To obtain these, we must avoid the error, too common, of striving after extreme brevity. Names of morbid states, the meaning of which is obvious, are, even if somewhat longer, to be preferred to shorter expressions, the meaning of which is obscure. We are apt to associate with brief obscure names the idea of definite diseases. But if we would gain and convey exact ideas of the diseases of the spinal cord, we must endeavor to substitute the idea of morbid processes for that of definite diseases.

A simple and convenient system of terminology lies close to hand. We have in the spinal cord, the two cornua of gray matter and the three columns, lateral, anterior, and posterior. In each of these situations the various morbid processes already described may occur, and we have only to combine the terms indicating the place and the lesion to have a system of terminology already partly in use, and which will altogether suffice for our present needs. Thus we may have a columnar or a cornual myelitis, hæmorrhage, sclerosis, degeneration, or growth. We may have, for instance, an "anterior cornual myelitis," or, for shortness (since we cannot yet diagnose posterior cornual diseases), a "cornual myelitis;" or we may have a cornual degeneration. For anterior cornual myelitis, the term "tephro-myelitis" has been proposed by Charcot, and "anterior polio-myelitis" by Kussmaul. The latter term has obtained wide currency, but its meaning is much less obvious than that of "anterior cornual myelitis." The simpler system of nomenclature I have employed throughout this lecture, and it has probably been readily intelligible, although unexplained.

V.—THE DISTINCTION OF FUNCTIONAL AND ORGANIC DISEASE.

No term is more loosely used in medical writings than "functional disease." It is commonly employed, in regard to the nervous system, as a designation for those affections in which no morbid changes have been hitherto discovered, even with the microscope, and from which recovery is possible. Unfortunately, we have no other general term for these cases, and it is to be regretted that we are compelled to make a positive term connote negative characteristics. Strictly speaking, a functional disease is one which consists in a disorder of function without any preceding alteration of nutrition. The affections of the nervous system which can be included under this definition are very few. Most of the examples of purely functional disturbance of one part are the result of disease in some other part of the nervous system. In most cases of so-called functional disease, we must assume changes in nutrition. In some, these changes are probably primary; in others they may be secondary to the disturbance of function.

Hysteria is the most frequent cause of symptoms that are within the range of the spinal functions, and not due to organic disease. Hysterical symptoms of spinal type usually have the form of loss of power in the legs, "hysterical paraplegia;" and one of the most frequent problems in diagnosis is the distinction of this from organic disease. But more than one morbid condition is included under the term "hysterical paraplegia." In the most characteristic form of the disease there is not any affection, even functional, of the spinal cord. There is loss of power over the legs, because there is in relation to the legs the peculiar ungeared state of the volitional centres, which is at the root of all pure hysteric palsy. There is no disturbance of the central functions of the cord. Reflex action from the skin, and myotatic irritability, are normal. The muscles do not waste, although they may become somewhat flabby from disuse. Retention of urine may occur, but there is never incontinence, nor is there any affection of the sphincter ani. Sensation on the legs is almost always normal. Thus, all objective indications of an affection of the cord are absent. It is necessary to establish this negative fact before any weight can be allowed to the positive indications of the hysterical nature of the case. These are rather suggestive indications than positive symptoms. One of them is the presence or history of other unequivocal symptoms of hysteria, globus, loss of voice, hysteroid convulsions, and the like. Another is the mode of onset. Hysterical paraplegia is often excited by some emotional shock, such as an alarm. But it rarely comes on instantly, or reaches a high degree in an hour or two, except when it immediately follows a severe emotional shock. Usually its development occupies several days or weeks, and it is often preceded by occasional attacks of momentary weakness in the legs. When a considerable degree of weakness comes on quite suddenly there have been usually such preceding transient attacks of slighter loss of power. A third indication is derived from the character of the weakness. It is very rarely absolute. Some power usually remains. The patient can move the legs, but cannot stand. Moreover, there are two peculiarities in the manner in which the legs are moved which are, perhaps, more significant than any other positive symptom. But the full power which can be exerted is not put forth at once. By continued effort and repeated urging much more force can be evoked ultimately than at first. Thus the patient, lying in bed, is told to raise the foot. She does so, slowly, for about six inches, and says she cannot get it any higher. Nevertheless she holds it there, and if urged ultimately raises it higher, a foot or eighteen inches from the couch. The second is that if the patient tries to exert force with a given group of muscles, the opponents of those muscles are put in action at the same time and in undue degree. Thus, if the knee is flexed, and the patient is told to try to extend it, the flexors are put in action, as well as the extensors, and prevent the movement. Resistance to passive movement is often hesitating and jerky. Occasionally a movement, at first steady, becomes modified, in a few minutes, by tremor, usually quick but variable, now fine, now coarser, and with quick sudden jerks. It has been mistaken for the spasm of disseminated sclerosis, but differs in not being uniformly wild and irregular. The tendency to contract other muscles than those which effect the desired movement, is often associated with an inability to relax the muscles voluntarily. This may prevent the knee-jerk being obtained. The flexors of the knee contract and prevent any movement when the patellar tendon is struck. I believe that it is this, and this alone, which has led to the assertion that the knee-jerk is sometimes lost in hysterical paraplegia, or that it varies, and can be obtained at one time and not at another. There is rarely anæsthesia. These several indications are separately of little significance; they derive value from their combination.

In another class of cases there is, in addition to the above symptoms, distinct indication of slight disturbance of the functions of the spinal cord itself. There is spinal tenderness, apparently due to a neuralgic state of the spinal membranes or ligaments. Pain in and about the spine is often complained of, and is increased by movement, but it is never unilateral, passing round one-half of the trunk, as in organic disease. There is a slight increase of myotatic irritability in the legs.

The knee-jerk is excessive; the rectus contraction can readily be produced by tapping the depressed patella. The front-tap contraction in the gastrocnemius may be obtained, and a "spurious foot-clonus" may occur when the ankle is passively flexed. This spurious clonus results, as already described (p. 233) from a voluntary and variable contraction in the calf muscles. There is never a regular persistent clonus, except when there is persistent hysterical contracture (to be presently described), a much rarer condition in hysterical paraplegia than in hysterical hemiplegia.

This morbid state of the central functions of the cord is probably, in some cases at least, the result of the complex combination of neuropathic tendencies, which is called hysteria. The clinical history of this disease affords many examples of the affection of lower centres, excited by emotional causes, but running a course to a considerable extent independent. Nutritional changes doubtless follow the disturbed function, and one case recorded by Charcot suggests that, after years, the changes in nutrition may attain the degree of visible structural alterations. But in many cases of hysterical paraplegia the change in nutrition is to a large extent, often altogether, removable by judicious treatment directed to the strengthening of the will, improvement of physical health, and the removal of the irritable state of the spinal cord.

In other cases the disturbance of the cord has an independent origin. Many weakly women, who are not hysterical, suffer from symptoms which indicate slight impaired action of the cord. They are bad walkers, soon tire, and when tired suffer pain in the spine. The muscles of the legs are flabby, sometimes very thin; the knee-jerk is excessive. This condition is often left after prostrating diseases, such as typhoid fever, repeated child-bearing, prolonged anæmia from any cause, and may be permanent. If such women are, or become, the subjects of hysteria, the spinal weakness determines the direction in which symptoms develop that are of ideal origin. It is often difficult to distinguish these cases from those last described. I believe, however, that those in which the disturbance of the central function of the cord is secondary to hysteria are for less common than those in which slight spinal weakness or pain in the back precedes the hysterical disturbance and determines it, and may endure after the volitional defect is removed. Often both may be due to a common cause, which impairs general health and lowers *nervous* tone.

These cases, in which volitional defect is combined with slight over-action of the spinal centres, give rise to considerable diagnostic difficulty, unless the complexity of the pathological condition is duly recognized.

In cases in which there is rigidity of the legs, the question arises whether there is true spasm or hysterical contracture. The problem is the more difficult, because in each condition the limbs are usually extended, and in each there may be a distinct foot-clonus. This may be developed in hysterical contracture, as it is in health when the calf-muscles are long contracted. But in the vast majority of cases attention to the following points will decide without difficulty the nature of the case. In hysterical contracture the muscular spasm is greatest at the extremity of the limb. The ankle-joint is extended, so that the dorsum of the foot is in the line of the tibia, and the foot is usually inverted. Any attempt to alter the position is resisted, and if the contracture is partially overcome the rigidity continues. It can be ultimately overcome, but considerable pain is produced. The contraction is constant. In spastic paraplegia the spasm is equal throughout the limb, or even greater at the proximal extremity. The effect is to fix both legs to the pelvis, so that if one is lifted the other is moved with it. The spasm is variable; at times it is slight, at others violent. It is excited by peripheral irritation. It is not only extensor in character, but is almost exclusively extensor. When the limbs are flexed they are supple, but as soon as they are extended they become rigid. If the ankle-joint is in extension, it is in consequence of actual shortening of the calf-muscles, which cannot be overcome. A clonus is usually obtained without difficulty, and is uniform. It is best obtained when the spasm is least. In hysterical contracture the clonus is usually variable, and best marked when the contracture is greatest. The attacks of so-called "spinal epilepsy," first tonic and then clonic spasm, excited by peripheral impressions, occur only in organic disease.

Hysterical symptoms are notoriously greatest when the patient is under examination. The patient walks best when she is unaware that she is being watched. The more attention is paid to a given symptom, by the patient or others, the greater it is. Under the influence of a strong motive, actions can be performed that cannot be effected by a simple volitional effort. Strong faradization, for instance, may make an hysterical patient move her leg when she cannot be made to move it by a voluntary effort. Care must be taken, however, not to mistake a reflex for a voluntary movement. Such a mistake frequently leads to error in diagnosis. In many cases of paraplegia, from disease of the dorsal cord, with entire loss of voluntary movement, a painful cutaneous impression will cause a flexion of the hip-joint, by which the leg is drawn up, closely resembling a voluntary movement. The observer must satisfy himself as to the character of the movement before he allows it to influence his diagnosis.

Other forms of so-called "functional paraplegia" are rare. Their most frequent causes are sexual excess and some morbid blood state, especially chronic alcoholism and gout. The loss of power is never absolute, rarely such as to prevent the patient walking, and varies from time to time. The legs "feel heavy;" they are readily fatigued, and are often the seat of abnormal sensations, tingling, formication, and the like. Sensation, reflex action, and myotatic irritability are all normal. The symptoms occur chiefly in adults and in males. The absence of indications of organic disease, the variations in the symptoms, and the recognition of the cause, usually render an accurate diagnosis practicable.

VI.—ILLUSTRATIONS OF DIAGNOSIS.

The following illustrations may render clearer the application of the methods of diagnosis. I will take, first, two cases in which the pathological diagnosis presented no difficulty, since both were cases of fractured spine, and complete paralysis of the legs occurred immediately on the accident, indicating direct damage to the cord by the displaced bone.

(1.) In the one case, that of a sailor, there was no irregularity of the spines to guide us as to the position of the injury, but this was clear enough from the symptoms. The legs were completely paralyzed, and all the muscles, when the patient came under observation, some months after the injury, were greatly wasted, faradaic irritability being extinct. This proved complete degeneration of the motor nerves arising from the lumbar enlargement. Sores had formed on the limbs and sacrum, indicating damage to the nerves which influence the nutrition of the skin. Sensation was at first lost, but afterwards returned as hyperæsthesia—suggesting initial damage and partial recovery of the nerves or tracts conveying sensation. The sphincters were powerless, and their condition was such as to indicate damage to, or separation from, their centres in the cord (see p. 243). From these symptoms we inferred damage by compression of the lower part of the lumbar enlargement, and of the nerves passing it. But what was the state of the dorsal region of the cord? Sensation above the groins was normal, but this does not exclude slight damage to the cord, since the impairment of sensation caused by slight damage may soon pass away. Here it was that the superficial trunk reflexes assisted us. We found that the epigastric reflex and the abdominal reflex were perfectly natural on each side, even in the lower part of the abdomen. The cremasteric reflex, however, was active on the right side, absent on the left; so that we had evidence that the dorsal cord was normal, and that the damage commenced at the 1st lumbar nerve, where the reflex loops were damaged on the left side and normal on the right, and that just below this point the damage was great. The patient died, and the autopsy revealed exactly the condition which had been diagnosed. The dorsal cord was uninjured, and so was the highest part of the lumbar enlargement; while its lower portion was split in two by a fracture, with displacement, of the 1st lumbar vertebra, which had also compressed the nerve-roots. Microscopical examination revealed, also, slighter mischief in the cord, extending as far as the upper part of the lumbar enlargement.

(2.) The other case is that of a girl, a patient in University College Hospital under the care of Mr. Heath. She fell off a house-top and became at once paraplegic. There were indications of damage to the bones about the 10th dorsal vertebra. The legs were completely paralyzed; but there was only slight wasting, the faradaic irritability of the muscles being preserved, although lowered; and reflex action was preserved. Hence it was inferred that the motor nerves were undegenerated, that the lumbar anterior cornua were not directly damaged, that the reflex loops were entire; in short, that the damage to the cord was at or above the highest part of the lumbar enlargement. There was loss of sensibility to pain in the legs, that to touch being perfect. Hence we inferred that the destruction of the cord was incomplete. This loss extended as high as the epigastrium—evidence of some damage to the cord as high as the origin of the 8th dorsal nerves. This was corroborated by the condition of the superficial reflexes of the trunk: the abdominal was lost on both sides; the epigastric was lost on the right side, but present on the left, indicating clearly the highest level of damage.

Thus there was evidence of affection of the cord from the origin of the 8th to that of the 11th dorsal pairs; but the symptoms did not show whether the damage was equal throughout this region. This information was, however, supplied by an examination of the faradaic irritability of the abdominal muscles. Above the umbilicus there was normal irritability; below the umbilicus it was gone—*i.e.*, the motor fibres of the 9th pair were undegenerated, their anterior cornua were undamaged, the fibres of the 10th pair, perhaps also the 11th pair, were degenerated, and the corresponding cornua probably damaged. As the lumbar enlargement was not directly damaged, we were able thus to limit with precision the considerable damage to the cord to the origin of the 10th, or 10th and 11th pairs. The loss of the epigastric reflex on the right side indicated that the damage to the cord on that side was greater than on the left. The subsequent progress of the case showed the significance of these indications. A month later the epigastric reflex returned on the right side, an indication of commencing recovery in the upper part of the damaged region. A few weeks later she gained some power of moving the left leg, but the reflex action became excessive in both legs, and the foot-clonus could be obtained. Now, four months after the injury, the abdominal reflexes are returning; a slight reflex can be obtained just above the groin, and above the umbilicus; none at or just below the umbilicus.[1]

In both these cases, thus, the information conveyed by the trunk reflexes was most important.

(3.) A man, aged twenty-eight, had suffered from weakness of the legs for more than two years. He was able to walk leaning forwards upon his sticks. His arms were unaffected. He could just flex the hips and extend the knees, but could not flex the knees, and scarcely the ankles. The right leg was the weaker. The legs were well nourished. Even as he entered the room, the clonic spasm at the ankle-joint, as the calf-muscles were put on the stretch, was conspicuous, and it was found that the knee-jerk was in great excess, and the foot-clonus could be obtained by the slightest pressure against the soles. A slight peripheral impression caused rigid spasm succeeded by clonic contraction as the stronger spasm passed lessened (spinal epilepsy, see p. 249). Thus the loss of power showed interruption to the motor path somewhere below the cervical enlargement. The preservation of the myotatic contractions, and the absence of wasting in the legs, showed the integrity of the lumbar reflex loops and gray matter, while the intensification of these contractions pointed to such over-action of the reflex centres as accompanies descending degeneration in the lateral columns.

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[1] Fifteen months after the accident, the left leg has regained considerable power: the right remains paralyzed. The muscle-reflex irritability has increased to spasm.

The next point was to search for any evidence of mischief beyond the motor tract. This was found in the fact that sensation to pain in the legs was perverted: the pain of a prick was felt, but in an abnormal manner. Sensation to touch was normal. Thus it was evident that somewhere the sensory tract also was interfered with to a slight degree. The same fact was indicated by a sense of constriction around the abdomen. The two symptoms pointed to a lesion extending beyond the motor tracts, *i.e.*, to a focal lesion, and the fact that the sense of constriction was around the lower part of the abdomen made it probable that the lesion was in the lower part of the dorsal region. The cutaneous reflexes of the trunk were then examined. On the left side the epigastric reflex and the abdominal reflex above the umbilicus were very active. Just below the level of the umbilicus the abdominal reflex was much lessened, and midway between the umbilicus and groin could not be obtained. On approaching Poupart's ligament it was again produced, and an impression here caused reflex flexion of the hip. Behind, the left dorsal reflex was active, the lumbar absent. On the right side, however, the abdominal reflex was extremely slight throughout, and the epigastric reflex could not be produced (although so active on the left side), and no lower dorsal or lumbar reflex was obtainable. Thus the reflex phenomena pointed to a very limited lesion on the left side, at the level of origin of the 11th dorsal nerve, while on the right side the more extensive loss indicated more extensive mischief in the right half of the lower dorsal cord, corresponding to the greater weakness of the right leg. This affection of the reflex in the left side corresponded to the position of the constricting band around the lower part of the abdomen. What was the nature of the lesion? Its onset was very gradual; the commencement was by a sensation of "numbness," followed, eight months later, by weakness. This extreme slowness pointed to degenerative changes—local "sclerosis." There was no bone disease, no history of syphilis; but the patient had been much exposed to wet two months before the onset, and we have seen that degenerative changes sometimes result from this cause.

(4.) A man, aged forty-eight, came under treatment for weakness in the right leg, chiefly marked in the movements of the foot. The corresponding arm was unaffected. Thus there was impairment of the motor conducting tract (lateral column) on the right side, somewhere below the cervical enlargement. Sensation was unimpaired; the sensory tract, therefore, undamaged. The nutrition of the legs was good; the plantar reflex ready and equal; the knee-jerk excessive in each leg; the foot-clonus and front-tap contraction obtainable in each. Hence the reflex loops and gray matter of the chief part of the lumbar enlargement were intact; the excess showed that there was probably lateral sclerosis descending from a lesion above, and that this existed on the left side as well as on the right. The lesion was thus evidently somewhere in the dorsal region of the cord. To learn its seat further, the higher superficial reflexes were examined. The cremaster reflex was distinct on the left side, not to be obtained on the right. The abdominal reflex was normal on the left side, but on the right it could not be obtained except just below the edge of the ribs. The epigastric reflex, excited from the side of the chest, was distinct on each side—as readily excited on the right side as on the left. Hence we had evidence, from the impairment of the reflex loops, that the cord was damaged on the right side, from the 8th dorsal nerve to the 1st lumbar. The excess of the myotatic irritability in the left leg suggests that the mischief had implicated slightly the left half of the cord, so as to lead to some descending degeneration, although not to any loss of the cutaneous reflexes. It is possible, however, that the descending degeneration in the left side of the lumbar enlargement, may have been due to the damage of fibres of the direct pyramidal tract on the right side, before their decussation to the left side.

So much for the anatomical diagnosis. The affection had come on acutely. In nine hours from the first symptom the leg was powerless, and it was five months before it recovered. The "acute" onset points to either a vascular lesion or inflammation. Causal indications were obscure. The man attributed his symptoms to a severe strain three days before, which he stated also caused "ulcerated bowels." The wide extent of the lesion in the right side of the cord, and its acute but not instantaneous onset, suggests the probability of a myelitis, not severe in degree, although acute in onset, rather than of hæmorrhage.

(5.) A young man, aged twenty-two, presented himself with weakness, wasting, and deformity of the left forearm. The muscles of the shoulder were normal. The upper arm muscles were rather smaller than those of the right side, but were of fair size and nutrition. The muscles of the forearm were greatly wasted, except the radial extensor of the wrist; the muscles of the thumb and little finger were much wasted, the interossei only slightly. The wasted muscles had lost faradaic and voltaic irritability, the condition being of long standing, and the muscular fibres probably totally degenerated. Sensation was perfect. The leg was not quite so strong as the other; nutrition normal; the myotatic irritability excessive in each leg; knee-jerk increased, and foot-clonus and front-tap contraction obtainable. Thus, we had evidence, from the muscular wasting, of a limited lesion in the left anterior cornu in the lower part of the cervical enlargement; from the impaired power in the leg, of slight damage to the motor tract for the leg; and from the excess of the myotatic contractions, of degeneration in the pyramidal tracts descending from above.

What was the nature of the lesion ? Its onset was sudden, nine years before. One morning he suddenly felt a pain in the back of his neck, then he found his arms and legs becoming weak, and this increased so rapidly, that in half an hour or so he was unable to move any limb, but there was no noticeable loss of sensation. Thus he lay for three weeks, and then the right arm began to regain power, next the right leg, and then the left leg, so that in two months he could walk. The wasting in the muscles of the left arm was very rapid. There was no causal indication. Thus there was a lesion of sudden onset, and therefore primarily vascular—thrombosis or hemorrhage—at first affecting a wide transverse area of the cord, and impairing all its functions at the spot, except conduction of sensation. The part slightly damaged soon recovered, but there remained an area of considerable damage in the left anterior cornu, and of slighter damage in the adjacent conducting tract to the leg. The focus of disease was no doubt the seat of the primary lesion; probably a local extravasation interfering with the opposite side of the cord by pressure.

(6.) The following case is somewhat complex in its indications, but is instructive as affording an illustration of the diagnosis of chronic meningitis. For an opportunity of seeing the patient, I am indebted to Dr. Russell, of Birmingham.

A man, aged forty-seven, complained of weakness of the legs, which was found, on examination, to be of irregular distribution. In both legs the muscles moving the hip-joint possessed good power. The flexors and extensors of the knee-joint were strong in the right, but very weak in the left leg. The flexors of the ankle in the left leg were rather weak, in the right were powerless; the extensors of the ankle were weak in both legs, but much weaker in the right than in the left. The muscles were wasted, and had lost faradaic irritability in proportion to their weakness, the voltaic irritability being preserved. The wasting was greatest in the extensors of the left knee, and flexors of the right ankle. In the latter faradaic irritability was gone. Sensation was normal, except in an area in the front of the left leg, where it was absolutely lost to both touch and pain. Plantar reflex normal; no foot-clonus; knee-jerk slight in the right leg, absent in the left.

The affection of nutrition and electrical irritability indicated disease in the anterior cornua, or in the motor nerves springing from them. By this the impairment of the knee-joint was also explained. The weakness was in proportion to the wasting; hence there was no reason to infer other disease than that interfering with nutrition. The diagnosis thus lay between a primary cornual disease, and damage to the nerve-roots by meningeal changes. The patch of anæsthesia in the left leg was in favor of the latter. It is rare that anterior cornual disease impairs sensation.

The mode of onset was then investigated to ascertain what light the order of the symptoms would throw on the seat, and their rapidity of development on the nature of the disease. The first symptoms commenced nine months before, and were sensory; soreness in the left leg, followed by shooting pains, sometimes in the big toe, sometimes in the calf, but confined to the left leg; these lasted for two months, and during that time the leg gradually got weak. After this similar pains were felt in the right leg, and this also became weak.

Pains of this darting character are usually due to irritation of the sensory nerve-roots; they are similar to those met with in locomotor ataxy, and are almost unknown in affections of the anterior cornua.

These pains, in conjunction with the patch of anæsthesia, rendered it highly probable that the mischief was outside the cord in the meninges —chronic meningitis with thickening, the nerve-roots being irregularly damaged by irritation and compression. Hence a careful search was made to ascertain if there were any trunk symptoms, throwing light on the disease. Inquiry elicited a history of shooting pains on the right side of the trunk, at the level of the epigastrium, with a unilateral sense of constriction. Some local hyperæsthesia was found, but no anæsthesia. This also pointed to local irritation of the posterior nerve-roots, higher up—to irregular meningeal irritation.

The causal influences were then ascertained. No immediate cause could be ascribed except general bad health. Chronic meningitis is most frequently due to bone disease or syphilis. There was no evidence of bone disease, but the patient had had a chancre twenty years before.

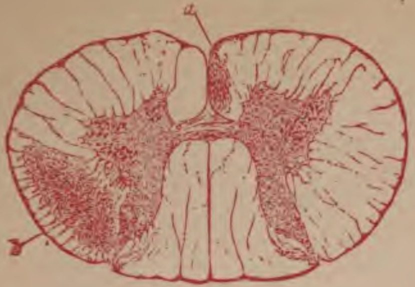
Thus the motor symptoms indicated either cornual or meningeal disease; the sensory symptoms pointed strongly to the latter, and the causal conditions, as far as they went, harmonized with the view. (The patient had had some sugar in the urine, which, except for rendering the prognosis worse, had little apparent bearing on the case.) He had taken iodide for a time, but without improvement. This did not, however, militate against the diagnosis for the following reason:—The meningeal change, though probably syphilitic, had caused damage to the nerves, which, descending as degeneration, had led to secondary changes in the muscles. The removal of the syphilitic change in the membranes would not at once restore the nerves. Their regeneration, if still possible, would be, of necessity, a work of time, perhaps of more time than had yet been allowed. Hence the patient was urged to persevere with the iodide, and mercury was added to it, and he was advised to continue the use of a voltaic battery to the muscles. A month later there was slight but distinct increase of power in the left thigh. I did not see him again, but a year later he was seen, incidentally, by Dr. Russell, who has been good enough to inform me that the man then said he was, and appeared to be, perfectly well. The result thus affords a strong confirmation of the diagnosis.

DESCRIPTION OF PLATE.

THE figures represent some of the more important lesions of the spinal cord. Although semi-diagrammatic, they have, with one exception, been drawn, with care, from actual sections. The exception is Fig. 2, which is after Charcot. It may be well to state, for the information of those unfamiliar with the process of microscopical examination of the nerve-centres, that when a section of the spinal cord is stained with carmine, the tint assumed by the different parts varies, and conveys important information. The gray substance stains much more deeply than the white, and the nerve-cells more deeply than the inter-cellular gray substance. Hence the gray matter appears like an H-shaped rose-colored area, in which the nerve-cells appear of a much deeper red. The white substance of the nerve-fibres does not stain, and although the axis-cylinders stain, they are not sufficient in bulk to give much color to the white columns. Connective tissue, however, stains very deeply, and the edge of the section (pia mater) is thus deep red, and so are the trabeculæ of connective tissue which extend into the white substance. Since the process of sclerosis consists in an atrophy of the nerve-fibres, and an increase in the connective tissue, areas so affected stain deeply in proportion to the intensity of the change, and its existence and degree may thus be rendered conspicuous even to the naked eye. The relative tint of the figures is nearly that of the sections from which they were drawn, all of which were stained with carmine. The letters indicating corresponding parts are the same in all the figures.

FIG. 1. *Descending Degeneration, unilateral.*—Section of spinal cord, cervical region, from a case of left hemiplegia due to disease of the right cerebral hemisphere. The two pyramidal tracts are degenerated, viz., the small "anterior pyramidal tract" (*a*), close to the anterior median fissure, on the right side of the cord; and the "lateral pyramidal tract" (*b*) in the opposite lateral column. This degenerated tract is seen not to extend up to the surface, being bounded by the so-called "cerebellar tract." (See p. 214.)

FIG. 2. *Descending Degeneration, bilateral.*—Section of spinal cord, dorsal region, below a point damaged by compression. Both lateral



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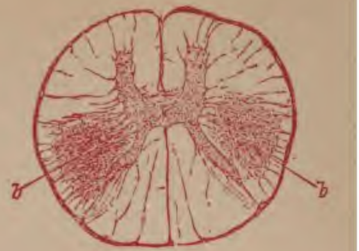

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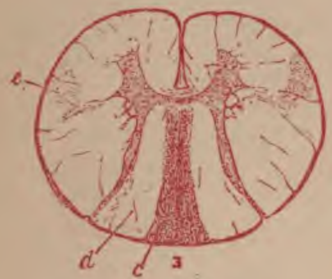

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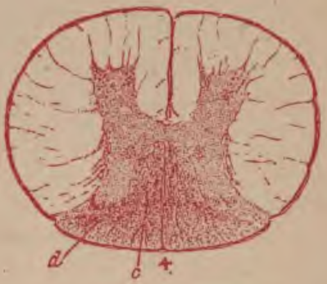

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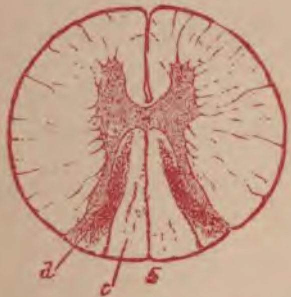

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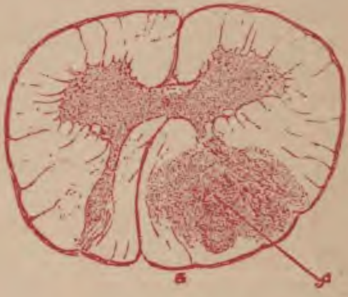

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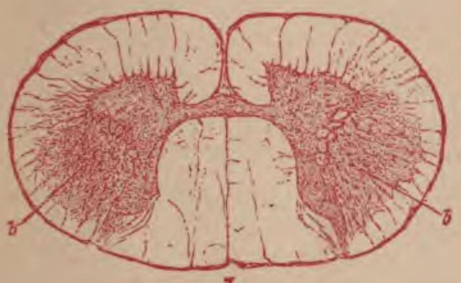

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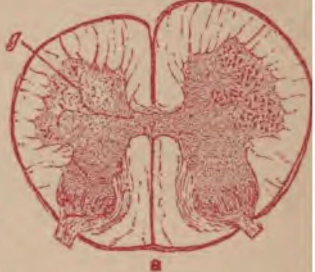

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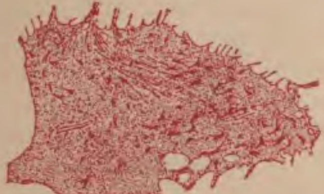

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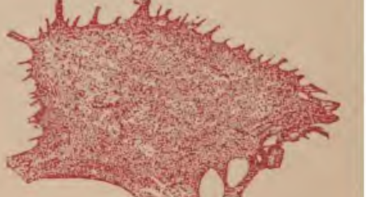

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pyramidal tracts (*bb*) are degenerated. There is no degeneration in the anterior pyramidal tracts, which had probably ceased (by decussation) above the level of the section, or may have been absent.

FIG. 3. *Ascending Degeneration.*—Section of spinal cord in dorsal region, from a case in which the lower extremity of the cord was crushed by a fracture of the spine. The postero-median columns (*c*) are densely sclerosed. The postero-external columns (*d*) are quite free from sclerosis. The pyramidal tracts in the lateral columns are seen to be also free from disease (compare Figs. 2 and 3), but just in front of each is a symmetrical area of slight degeneration (*e*). (See p. 216.)

FIG. 4. *Posterior Sclerosis, Locomotor Ataxy.*—Section at the level of the first lumbar nerves. The posterior columns are densely sclerosed throughout their entire extent. The remaining white columns and anterior cornua are healthy.

FIG. 5. *Sclerosis of Postero-external Column* (*posterior root-zone*), *Locomotor Ataxy* (from a section prepared by Prof. Pierret, of Lyons). —A dense band of sclerosis occupies the postero-external column (*d*), through which the posterior nerve-roots pass. The postero-median columns (*c*) are free from sclerosis. The bands of sclerosis are narrow, probably from the contraction of the tissue, since, from the position of the limiting septum, they appear to occupy the entire width of this column. The patient suffered from well-marked locomotor ataxy.

FIG. 6. *Syphilitic Growth in Posterior Column.*—Section through the spinal cord, cervical region, of a man who died from syphilitic disease of the brain. A growth (*f*) occupies the right postero-external column, and has enlarged it to three times the normal size, displacing the posterior median septum to the left. The growth has invaded the right posterior cornu, and extended a little way beyond it into the lateral column. It caused inco-ordination, and partial loss of sensibility, in the right arm.

FIG. 7. *Anterior Cornual Degeneration.*—Section of spinal cord, cervical region, from a patient suffering from progressive muscular atrophy. The gray substance of the anterior cornua is degenerated and irregularly translucent, the nerve-cells having disappeared (compare also Figs. 9 and 10). The lateral columns (pyramidal tracts) are also sclerosed. The posterior columns are healthy.

FIG. 8. *Anterior Cornual Myelitis* (*Infantile Paralysis*).—Section of spinal cord, lumbar enlargement, from a case of old infantile paralysis of the left leg. The whole left half of the cord is smaller than the right. The left anterior cornu is shrunken, and presents evidence of previous inflammation. The tissue is degenerated and translucent, containing large vessels. All the motor nerve-cells, so conspicuous on the other side, have disappeared.

Fig. 9.—Normal anterior cornu, cervical region, showing numerous multipolar nerve-cells.

Fig. 10.—Anterior cornu, same position, from a case of progressive muscular atrophy. All the nerve-cells have disappeared; minute shrunken corpuscles here and there are probably their remains. The gray matrix, instead of being uniform, is irregular, translucent at some spots, unduly dense, from sclerosis, at others, especially near the edge of the cornu.

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